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PRELIMINARY INVESTIGATION OF EFFECTS ON THE ENVIRONMENT OF BORON, INDIUM NICKEL, SELENIUM, TIN, VANADIUM AND THEIR COMPOUNDS

VOLUME IV - SELENIUM

VERSAR, INCORPORATED

PREPARED FOR
ENVIRONMENTAL PROTECTION AGENCY

AUGUST 1975

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VOLUME IV

SELENIUM

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PRELIMINARY INVESTIGATION OF EFFECTS ON THE ENVIRONMENT OF BORON, INDIUM NICKEL, SELENIUM, TIN, VANADIUM AND THEIR COMPOUNDS

Volume IV

Selenium

Contract No. 68-01-2215

Project Officer
Farley Fisher

Prepared for

Office of Toxic Substances
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Washington, D.C. 20460

August 1975

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SELENIUM

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Volume IV

Preliminary Investigation of Effects on Environment of Selenium and Its Compounds

FOREWORD

This is Volume IV of a series of six reports on the environmental effects of boron, indium, nickel, selenium, tin, and vanadium and their compounds. The information is based on literature reviews, direct contact with representatives of companies involved in the production or use of the materials, and consultation with knowledgeable individuals from industry, academic institutions and the Federal Government.

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I. PRODUCERS, SITES AND COSTS

A. Producers and Sites

Table 1 lists commercially significant chemical products and the companies involved. For this study significant is defined as production exceeding 1/2 metric ton or $1,000 value. Other materials may also be included in the discussion because of their unusual properties, such as toxicity, or their anticipated future significance.

Selenium Products and Producers

Table 1.
(1,2)

Chemical	Producer: Company, subordination	Location
Ammonium selenite	City Chem. Corp.	Jersey City, N.J.
Barium selenate	City Chem. Corp.	Jersey City, N.J.
Barium selenite	Fairmont Chem. Co., Inc.	Newark, N.J.
	Filo Color and Chem. Corp.	Newark, N.J.
Cadmium reds (cadmium selenide lithopone)	Hercules Inc., Coatings & Specialty Products Dept.	Glen Falls, N.Y.
	H. Kohnstamm & Co., Inc., Gen. Color Co.	Newark, N.J.
	SCM Corp., Glidden-Durkee Div., Pigments and Color Group	Baltimore, Md.
	Smith Chem. & Color Co., Inc.	Jamaica, N.Y
Cadmium selenate	City Chem. Corp.	Jersey City, N.J.
Cadmium selenide	Allied Chem. Corp. Specialty Chems. Div.	Marcus Hook, Pa.
	Kewanee Oil Co., Harshaw Chem. Co., Crystal & Electronic Prod. Dept.	Solon, Ohio
Calcium selenate	City Chem. Corp.	Jersey City, N.J.
Carbon diselenide	Strem Chems. Inc.	Danvers, Mass.
Chromium selenium	Kawecki Berylco Indust., Inc.	Boyertown, Pa.

Selenium Products and Producers

Table 1. (cont).

Chemical	Producer: Company, subordination	Location
Cobaltous selenate	City Chem. Corp.	Jersey City, N.J.
Cobaltous selenite	City Chem. Corp.	Jersey City, N.J.
Cupric selenite	City Chem. Corp.	Jersey City, N.J.
	Fairmont Chem. Co., Inc.	Newark, N.J.
Cuprous selenide	The Shepherd Chem. Co.	Cincinnati, Ohio
Hydrogen selenide	G.D. Searle & Co., Will Ross, Inc.	Cucamonga, Calif.
	Matheson Gas Products	East Rutherford, N.J. Gloucester, Mass. Joliet, Ill. La Porta, Tex. Morrow, Ga. Newark, Calif.
Potassium selenocyanate	City Chem. Corp.	Jersey City, N.J.
	Filo Color and Chem. Corp.	Newark, N.J.
Selenic acid	City Chem. Corp.	Jersey City, N.J.
Selenium (Metal)	Alloychem, Inc.	New York, N.Y.
	American Smelting & Refining Co.	New York, N.Y.
	Atomergic Chemetals Co.	Carle Place, N.Y.
	Gallard-Schlesinger	Los Angeles, Cal.
	Bram Metallurgical Chem. Co.	Philadelphia, Pa.
	City Chem Corp.	New York, N.Y.
	Electronic Space Products, Inc.	Los Angeles, Cal.
	Fairmont Chem. Co., Inc.	Newark, N.J.
	Goldsmith, DF Chem. & Metal Corp.	Evanston, Ill.
	Kewanee Oil Co., Harshaw Chem. Co.	Cleveland, Ohio
	Hercules Inc., Drakenfeld Colors	Washington, Pa.
	Indussa Corp.	New York, N.Y.

Selenium Products and Producers

Table 1. (cont).

Chemical	Producer: Company, subordination	Location
Selenium (Metal) (cont)	Kawecki Berylco Ind., Inc.	New York, N.Y.
	Phelps Dodge Refining Corp.	New York, N.Y.
	Robecko Chems. Inc.	New York, N.Y.
	Sylvan Chem. Corp.	Englewood Cliffs, N.J.
	Var-Lac-Oid Chem. Co.	Elizabeth, N.J.
	Ventron Corp., Alfa Products	Beverly, Mass.
Selenium dioxide	Apache Chems., Inc.	Rockfield, Ill.
	Fairmont Chem. Co., Inc.	Newark, N.J.
	Filo Color and Chem. Corp.	Newark, N.J.
	Kawecki Berylco Indust., Inc.	Boyertown, Pa.
Seleium monochloride	City Chem. Corp.	Jersey City, N.J.
Selenium oxychloride	Fairmont Chem. Co., Inc.	Newark, N.J.
Selenious acid	City Chem. Corp.	Jersey City, N.J.
	Fairmont Chem. Co., Inc.	Newark, N.J.
	Filo Color and Chem. Corp.	Newark, N.J.
Sodium selenate	City Chem. Corp.	Jersey City, N.J.
	Fairmont Chem. Co., Inc.	Newark, N.J.
Sodium selenite	City Chem. Corp.	Jersey City, N.J.
	Fairmont Chem. Co., Inc.	Newark, N.J.
	Filo Color and Chem. Corp.	Newark, N.J.
Zinc selenide	City Chem. Corp.	Jersey City, N.J
	Kewanee Oil Co., Harshaw Chem. Co.	Solon, Ohio
Zinc selenite	City Chem. Corp.	Jersey City, N.J.
	Fairmont Chem. Co., Inc.	Newark, N.J.
	Filo Color and Chem. Corp.	Newark, N.J.
	The Shepherd Chem. Co.	Cincinnati, Ohio

B. Costs

The 1972 producers' price for commercial and high-purity selenium was $19.80 and $25.30 per kilogram. The dealers' price for commercial grade selenium was $19.80 to $20.50 in the beginning of the year, slumped below $19.50 in August, and was back to $19.80 at the year's end.[3]

Since most of the commercial selenium compounds are produced from selenium metal, their prices are primarily based on the selenium prices above plus processing costs.

C. Physical Properties

The major materials covered in this report are selenium metal and selenium dioxide. Physical properties of these materials are presented in Table 2.

Table 2

Physical Properties of Selenium and Selenium Dioxide[5]

	Specific Gravity	Melting Point °C	Boiling Point °C	Solubility in water g/100cc @ °C
Selenium	4.82	220	688	insoluble
SeO_2	3.95	340-350	sublimes 317	38.4^{14} 82.5^{65}

II. PRODUCTION

A. Producers

1. In 1972 primary selenium production was at four major electrolytic copper refiners:[3]

U.S. Metals Refining Co. American Metal-Climax	Carteret, N.J.
American Smelting & Refining Co.	Baltimore, Md.
International Smelting & Ref. Co. Anaconda Co.	Perth Amboy, N.J.
Kennecott Copper Corp.	Garfield, Ut.

The Inspiration Consolidated Copper Co., Magna Copper Co., and Phelps Dodge Corp. transferred crude materials containing primary selenium from their copper refineries to the above plants.

2. Selenium is recovered by domestic secondary refineries from purchased electronic scrap.

B. Quantities

1. Production

In 1972, 335,000 kilograms of primary selenium were produced; secondary selenium production is estimated at 13,500 kilograms. Considerable selenium domestic scrap was reused by manufacturers after outside reprocessing. Some selenium-containing material was also shipped to foreign plants for recovery.[3]

In August, 1971, Congress authorized the disposal of 210,000 kilograms of selenium held in national stockpile. In 1972, 7,290 kilograms of selenium from the stockpile were sold or exchanged.[3]

2. Production trends

Table 3 shows a trend in the U.S. production of selenium together with the amounts imported for domestic consumption. The variations are caused in part by trends in the copper industry, on which selenium production depends. In fact, the United States was the world's leading selenium producer prior to the

1967-68 copper strike. The imports for consumption do not offset production changes.

Table 3 (3,5)

Selenium and Import Trends, metric tons selenium

	1968	1969	1970	1971	1972
Production	287	565	455	298	348
Imports	210	248	205	179	195

C. Process

1. Selenium recovery

Copper refinery anode slimes contain from 3 to 28 per cent selenium. They provide the greater part of the U.S. and world production of selenium. The sludge is aerated with dilute sulfuric acid to separate the copper. In one process for recovering the selenium, the slime residue is heated with soda ash and silica and oxidized to the dioxide by blowing air through the melt. Some of the selenium dioxide sublimes and is collected in water or aqueous alkali in a wet scrubber system. Then sodium hydroxide alone or mixed with sodium nitrate is added to the selenium dioxide to form sodium selenite. The melt solidifies on cooling, and is crushed. At this point the selenium salt is leached with cold water. Up to this point, tellurium, which is present in the anode slimes along with selenium, is carried along with the selenium in the processing. The alkaline leach liquor is neutralized with sulfuric acid, precipitating the bulk of the tellurium. The hydrated dioxide with selenous acid remains in solution. Fairly pure (99.5%) selenium is precipitated with sulfur dioxide. The treatment followed next depends on the impurities.(6)

An alternative procedure is to evaporate the leach liquor from the roasted slime to dryness. Coke reduces the selenium salts (selenites or selenates) to the selenide. The sodium

selenide is dissolved in water and treated with air to precipitate pure selenium:[6]

$$Na_2Se + H_2O + \frac{1}{2}O_2 \longrightarrow Se + 2NaOH$$

The details of the process used by industry are not available because of proprietary considerations. Industrial processes are complicated and generally tailored to a special mix of recoverable valuable components. The components include copper, silver, gold, tellurium, and metals of the platinum group. Some information has been published about the Kennecott Copper Corporation operation at Garfield, including qualitative information of resulting environmental contamination from this operation.

In the process used by the Kennecott Copper Corp. the decopperized slimes are smelted in a rotary kiln with sodium bisulfate, or a mixture of sodium sulfate and sulfuric acid. The selenium dioxide formed is volatized and recovered in a scrubber-Cottrell system. The selenium-free residue, a fluid soda slag, is discharged into water or granulated and water-leached. The crude selenium is dissolved in aqueous sodium sulfite, and the product treated with sulfuric acid.

$$Na_2SeSO_3 + H_2SO_4 \longrightarrow Na_2SO_4 + Se + SO_2 + H_2O$$

The selenium formed is distilled several times, discarding the first and last fractions. It is then formed into a shot and sold as the high-purity grade.[7]

2. Selenium compounds

a. Ferroselenium

Ferroselenium is prepared from a fused mixture of powdered iron and powdered selenium.[7]

b. Selenium dioxide

Selenium dioxide is an intermediate in the extraction of selenium from anode slimes. Commercial selenium dioxide is prepared either by the catalytic oxidation of selenium with air or treatment with nitric acid and evaporation.[7]

c. Selenium oxychloride

Selenium oxychloride may be prepared by chlorinating a mixture of selenium and selenium dioxide dry or suspended in carbon tetrachloride.[7]

III. USES

A. Selenium and its Compounds

A breakdown of the estimated use pattern for selenium in 1972 is shown in Figure 1. Selenium consumption in 1972 increased about 10 per cent over that of the previous year. A detailed summary of current selenium usage is presented in Table 4.

B. Future and Potential Uses

There are suitable substitutes for selenium in many of its applications. For many electronic applications germanium or silicon can be used. In the chemical, rubber, and steel industries, sulfur and tellurium are possible substitutes. In copying devices, zinc oxide and certain dyes and organic materials can serve as photo conductors. Under current technology, however, only selenium is suitable in reusable photosensitive plates.[5]

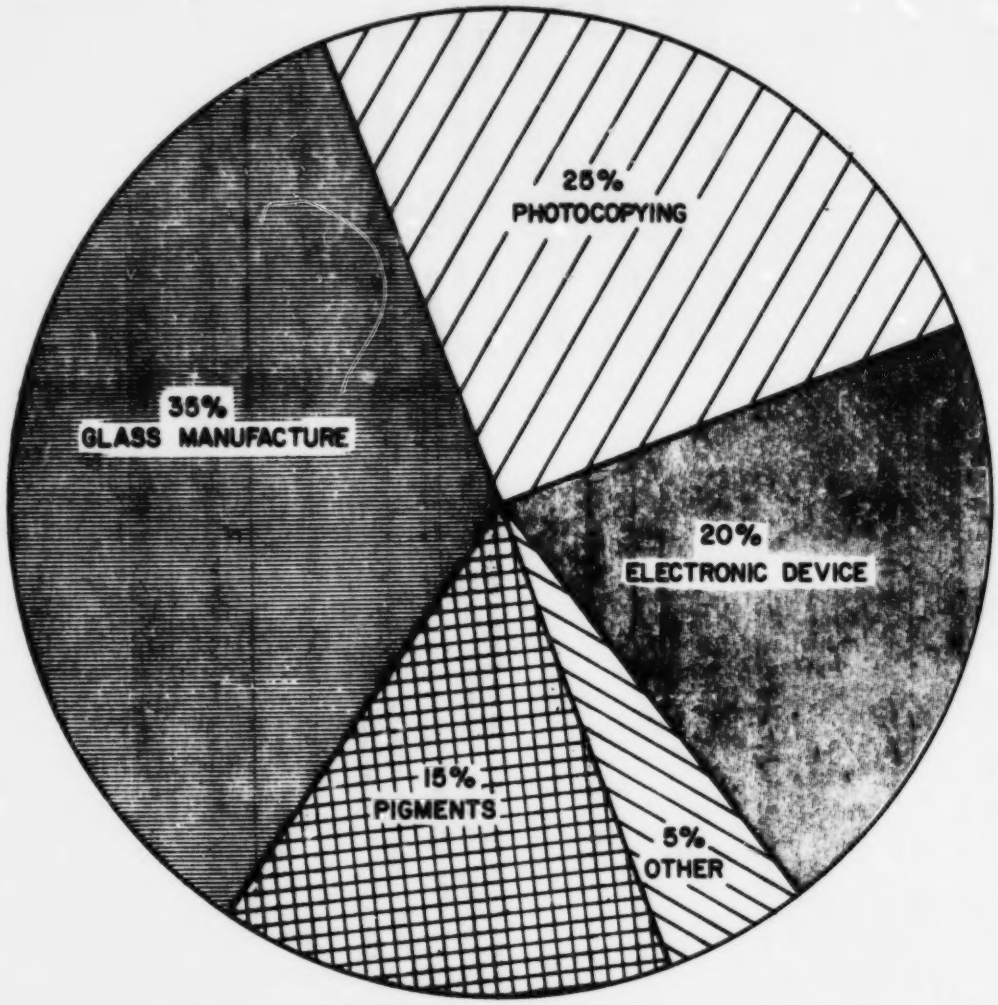

Figure 1
Selenium uses

Table 4.

Table of Uses
(3)

Compound	Use	Purpose	Comments
Selenium and its Compounds	Glass manufacture	Small amounts of selenium added to glass melts to neutralize the green color caused by iron.	Amount of selenium used for this purpose increased 15% from 197' to 1972
		Large amounts used to produce gray and bronze window glass that reduces glare and heat transmission	
		Used in manufacture of red and amber-colored glass for signals and decorative uses	
	Photocopy Machines		The greater use of copying machines caused only slight increase in selenium usage from 1971 to 1972 due to more efficient use of selenium and the reclaiming of domestic scrap
	Electronic devices		Industry expansion was offset by more efficient use of selenium compoun
	Pigments	Bulk of pigment was required for production of "cadmium red" (cadmium sulfoselenides)	Amount of selenium used for this purpose increased appreciably from 1971 to 1972
	Other	Small amounts used as additive to poultry and swine feed	Produces significant beneficial effects

IV. CURRENT PRACTICE

A. Handling and Transportation

Commercial elemental selenium is relatively inert and may be handled without special precaution. This is also true for stable metal selenides, such as copper and lead. All other selenium compounds should be treated with caution since some of these compounds can enter through the lungs or skin and may affect the body organs. This includes the reactive selenides, the volatile and soluble compounds, and particularly hydrogen selenide and organics.

Selenic acid with a melting point of 58C, is classified as a corrosive liquid, and transport containers must carry a white label. Cargo aircraft cannot accept more than 25 liters of selenic acid and passenger aircraft transport is prohibited. When handled as a solid, selenic acid requires a poison label. Quantities of solid selenic acid up to 25 kilograms can be shipped in passenger aircraft and 95 kilograms can be shipped in cargo planes.(8)

One manufacturer uses the following containers to ship selenium:(9)

(a) Selenium (commercial grade, powder of 80, 140, 200 and 325 mesh, selenium analysis - 99.7%) - packaged in 45 kg pail.

(b) Selenium (high-purity, pellets of 1/8" diameter, analysis 99.99% selenium minimum) - packaged in 55 kg fiber drum containing four 13.5 kg fiber polyethylene-lined containers.

B. Disposal

Selenium metal and selenium compounds must be disposed of in closed containers.(10)

V. ENVIRONMENTAL CONTAMINATION

A. From Use

a. Glass

Selenium, or in some cases sodium selenate, sodium selenite, or barium selenite is added to glass in small amounts to decolor it or in larger amounts to color it. Material may be lost to the environment when the raw materials are handled. Salts of selenium, which are soluble, serve as an example of this. Mixing ingredients for glass making, especially in batch processing, is done with dry materials causing some loss by dusting. A large amount of the elemental selenium used is volatized during melting as was confirmed by stack analysis. The glass industry estimates that 184 metric tons of selenium were emitted into the atmosphere in 1969.(11) Except for possible local concern, this should not pose an environmental problem. Data is not available on selenium loss to the environment via other channels. Considering the nature of the processes involved, however, this figure should be insignificant.

b. Photocopying

For the most part the photocopy processes of charging, exposure, image development, image transfer, image fixing, and photoceptor cleaning take place in an enclosed environment, presenting little chance for contamination. The exception is in the manufacture of photocopy equipment, where selenium is lost to the atmosphere in the vacuum plating operation. The industry estimates that about one kilogram per metric ton of selenium processed or around 135 kilograms in 1972 escaped into the atmosphere.(11)

c. Pigments, Chemicals and Miscellaneous

The pigments are dispersed in liquid media, applied by painting, spraying, screen printing or dipping, and fired at 480 to 870 C. Bag filters are used to control emission to the atmosphere, and losses to the environment via other routes are very small. Among other uses of selenium, its use as an insecticide, particularly for carnations and crysanthemums, allows small amounts to get into the environment.(11)

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B. From Production

Selenium recovery in the United States is principally a by-product of copper refining. The environmental contamination from selenium is closely related to the mining and milling of copper-bearing ores during the recovery of selenium. Unfortunately, analyses of the wastes are not available.

A major producer of selenium and precious metals by recovery from the copper-bearing ore has recently started analyzing waste streams. The analyses cover a long list of elements including selenium and is complicated by the extremely small concentration levels. Some qualitative information is provided on losses of selenium to the environment. Selenium was found in a number of waste streams within the operation. The precious metals recovery is a probable source of loss of selenium. In the selenium recovery process there are several points where selenium vapor may be lost. Besides the melting operation and drying oven, which represent small potential losses of selenium, the bisulfate fusion is of great concern. In the original installation a wet scrubber was installed on the exhaust side followed by a electrostator precipitator. In 1971 new hooding and wet scrubbers followed by electrostatic precipitators were installed at all vapor discharge points. This eliminated visual emissions to the atmosphere, but no analyses of the exhausts are available.

Selenium is also present in the very hot exhaust gases from the reverberatory furnaces and converters in the copper smelter. This is revealed by chemical analysis of particulate matter collected in the balloon flues and electrostatic precipitators through which the exhaust gases flow. Selenium is also found in waste water stream from the selenium recovery plant and from the wet scrubbers ahead of the sulfuric acid plant. Selenium may appear in the water used in leaching metal values from waste dumps.

C. From Inadvertent Sources

A survey was made in a representative U.S. town of the amount of selenium in solid waste. The objective was to determine the extent to which the handling, processing, and disposal of solid waste contributed selenium to the environment. Emphasis was placed on emission of selenium as a result of incinerating solid waste. The highest concentration observed was 14.5 micrograms per gram of particulate matter collected in a stack emission sample. Maximum values of 0.014 and 0.023 milligrams per liter were observed for an incinerator residue quench water and an incinerator fly ash quench water sample, respectively.(18)

During 1971 coal samples were analyzed for several elements including selenium. The average selenium content was 8 ppm for 15 samples of coal taken from various parts of the United States. During the combustion of coal, the selenium is discharged with the ash, of which fly ash averages about 65 per cent of the total. In 1969 the total consumption of bituminous and anthracite coal consumed was 469,000,000 metric tons. Based on this figure, 90 per cent application of controls, and 85 per cent efficiency of controls, the selenium emissions were calculated to be 573 metric tons.(11)

Until recently, data on the selenium content of oil was almost non-existent. Since 1971, however, analyses have been made for the Office of Air Programs of the Environmental Protection Agency. The average selenium content was 0.4 ppm for 10 samples of foreign and domestic crude oil. Assuming the above figure and the combustion of 76 x 106 cubic meters of residual fuel oil, the selenium emissions to the atmosphere amounted to 30 metric tons for 1969.(11)

D. Amount Entering the Environment

Probably the major source of selenium in the environment is the weathering of natural rocks. This apparently introduces much more selenium into the waters and the atmosphere than the total of sources attributed to man. In addition, selenium enters food chains directly through plants grown on seleniferous soil. These factors are discussed in detail in Section IX.

Sources attributal to the activities of man, as described earlier in Section V, can account for approximately 3,500 metric tons of selenium per year entering the atmosphere (about 900 metric tons per year) and landfills or dumps (about 2,600 metric tons per year), based on:

Source	Amount Entering Environment per Year
Glass Manufacture	200 kkg (mostly atmosphere)
Fuel Oil Combustion	30 kkg (atmosphere)
Coal Combustion	573 kkg - to atmosphere
	2,500 kkg - to land
Other	200 kkg
Total	3,503 kkg

VI. MONITORING AND ANALYSIS

A. Monitoring

There are no data available on the monitoring for selenium of air or water.

B. Analysis of Selenium

In most methods used for the analysis of selenium, the selenium must be in solution as the quadrivalent ion. It is then either reduced and precipitated as the element or measured volumetrically by oxidation to the hexivalent state, or by iodimetry. Selenium can also be determined polarographically. The only specific spot test for selenium depends on its catalytic action on the reduction and decoloration of methylene blue by alkali sulfides. A drop of the solution to be tested is added to a few crystals of sodium sulfite, followed by a little sodium sulfide solution, and then the methylene blue. The sensitivity is 0.08 micrograms.[19]

C. Analysis of Selenium in Urine

Urine is digested in a sulfuric-nitric acid mixture followed by distillation with $HBr-Br_2$ solution which separates selenium as $SeBr_4$. Sulfur dioxide precipitates selenium which is filtered off and estimated by an iodine thiosulfate titration.[20]

D. Analysis of Selenous Acid

This spectrophotometric method for the determination of submicron quantities of selenous acid was developed in 1971. The procedure is based on the oxidation of hydroxylamine hydrochloride to nitrous acid by selenous acid followed by the subsequent coupling of the diazonium salt with N(1-naphthyl)-ethylenediamine dihydrochloride. Optimum conditions have been established. The range of determination is 0.01 to 0.20 mg Se^{IV} per liter. The method is simple, sensitive, and reproducible with no common interferences which cannot be easily obviated.[21]

E. Detection of Selenium in 10 part per billion range

The current level of selenium tolerated in Illinois public waters is 10 parts per billion (0.01 mg/l) which is beyond the capability of classical analytical methods due to limitations of volume of sample, time of analysis, or removal of interfering substances. A fluorometric method was developed to analyze the selenium by the Analytical Research Department of Abbott Laboratories, North Chicago, Illinois. Elemental selenium is converted to selenious acid by the action of a bromine-bromide redox buffer. Selenious acid reacts with 2, 3-diaminonaphthalene in acid solution to form the strongly fluorescent naphtho-[2,3-d]-2-seleno-1,3-diazole. Selenates are not reduced to selenites under these conditions, hence the method is specific for elemental selenium and selenium in the four valance state. Plots of fluorescent intensity vs concentration in the region of 0.005 to 0.2 µg selenium (IV) are linear and practically free from reagent interference at excitation frequency of 366 nm and a fluorescent emission minimum of 522 nm.[22]

VII. CHEMICAL REACTIVITY

A. Environmental and Use Associated Reactions

The bulk of the uses of selenium are of non-chemical nature, so chemical reactivity plays a limited role.

B. Aspects with Biological Implications

The exhalation of volatile selenium compounds by plants, animals, and microorganisms is a minute source of selenium to the atmosphere. Selenium may be acted on by bacteria in nature to produce highly poisonous compounds,[23] such as dimethyl diselenide, given off by certain plants, and dimethyl selenide, given off by microorganisms and animals.[24]

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VIII. BIOLOGY

A. Absorption and Excretion

1. Humans

Selenium is absorbed through the lung, alimentary tract, and hands. Excretion is via respiration, urine, feces, milk, and perspiration. The concentration of selenium in the urine may reflect selenium exposure. The normal range for selenium concentration in the urine of healthy male adults is 0.10 to 1.50 ppm.[25] One study of farm families attempted to correlate urinary selenium with selenium levels in the diet. Table 5 shows a trend of increased selenium in the urine of individuals consuming foods high in selenium content. Except for one individual, all the urinary selenium concentrations fall within the normal range.[12]

A study of Oregon school children gave good correlation between selenium in foods, urinary selenium excretion, and prevalence of dental caries.[12]

2. Other Mammals

Orally ingested selenium is absorbed by the small intestine. Excretion is mainly through the urine, but also in the feces, perspiration, and exhaled air. The solubility of the selenium compound and the ratio of sulfur to selenium determine the rate of selenium absorption.[26] Intestinal transport of sulfur-35 methionine and selenium-75-selenomethionine apparently proceed via the same active mechanism in everted hamster intestinal sacs.[27] Selenite and selenocystine are absorbed by a different mechanism. Their sulfur analogs do not affect their absorption.[28]

Orally administered selenium was not absorbed from the rumen or abomasum (gastric stomach) of sheep or from the stomach of swine. In both species, selenium was secreted into the duodenum, due to its presence in pancreatic juice bile, and intestinal secretions.[29] Only 35 per cent of ingested selenium was absorbed by the sheep, compared with 85 per cent of the same dose absorbed by swine when rations containing 0.35 and 0.50 ppm selenium, respectively, were consumed. Monogastrics absorb selenium with a higher efficiency than ruminants, perhaps because ingested selenite could be converted to in-

Table 5

Relation of Urinary Selenium to Food Selenium

Family No.	Urinary selenium (ppm)	Food selenium (ppm)				
		Milk	Eggs	Meat	Vegetables	Cereal grains
97	0.25, 0.27, 0.32	Trace	0.57	—	Trace	Trace
51	0.20, 0.27	—	1.35	1.60	0.36	1.90
22	0.20, 0.21	0.36	1.40	—	0.41-0.74	Trace
113	0.20, 0.20, 0.24	0.25	1.45	—	0.30-0.82	0
52	0.26	0.25	0.32	—	Trace-0.58	—
83	0.13, 0.38, 0.40	0.34	—	2.19	—	—
27	0.29, 0.56	0.22	—	2.22	—	—
76	0.43, 0.73	0.35	4.08	3.30	0.27-1.05	—
107	0.94	0.39	3.65	—	Trace-0.18	—
47	0.70, 0.80, 0.98	0.57	3.08	—	0.23-2.04	3.30
74	1.03, 1.10	Trace	4.12	—	—	0.45-1.00
78	1.00, 1.14	0.36	5.04	—	1.03-17.80	3.60
16	1.05, 0.36, 1.33	1.14	—	—	2.42	2.50-18.80
19	1.24, 1.98	1.27	—	8.00	1.26	4.20-10.00

soluble forms by rumen microorganisms. Elemental selenium is formed from both selenite and seleno-amino acids in the gastrointestinal tract of ruminants, indicating both reductive and oxidative reactions taking place.[28]

Selenium intake, retention, and excretion were studied in rats (monogastrics) fed different forms and concentrations of selenium for 70 days in a corn torula yeast diet.[30] The apparent absorption of selenium averaged about 80 per cent of intake for all diets. The selenium balance in the rats was examined at day eight (trial I) and day 50 (trial II). The daily selenium intake in micrograms increased from day eight to day 50, but the 72 hour selenium retention at day 50 was reduced to less than half the retention at day eight. The percentage of fecal excretion of the selenium intake did not change from day eight to day 50.

At both times, selenite-fed rats excreted a higher percentage of selenium in the feces than selenomethionine-fed rats. The percentage of urinary excretion of selenium intake was more than doubled at day 50 compared with day eight. In rats fed 0.146 ppm selenium as selenite, the percentage of selenium excreted in the urine increased from 6.4 per cent to 32.5 per cent from day eight to day 50. The selenium intake in this latter group of rats was highest of all groups. The increased urinary excretion and decreased selenium retention indicate that perhaps selenium equilibrium was reached.

In lambs (ruminants) fed torula yeast diets containing 0.014 to 5.014 ppm selenium as sodium selenite for 97 days, the excretion of either intravenously or orally administered selenium-75-sodium selenite in the feces, urine, and respiration was determined during a 12-day period.[31] The fecal excretion, expressed as a percentage of administered dose of radioactivity, was about five per cent in lambs intravenously dosed, and about 20 to 25 per cent in orally-dosed lambs. (This latter is comparable to the percentage of oral selenium intake excreted in the feces of rats). The percentage of fecal excretion was unaffected by dietary levels of selenium indicating that the amount absorbed depends upon the amount taken in. Both urinary and volatile

selenium excretion increased with increasing dietary level of selenium. The effect on urinary excretion was most pronounced, ranging from less than 10 per cent of the orally or intravenously administered radioactivity in rats fed 0.14 ppm selenium to more than 40 per cent of the oral dose, and 60 per cent of the intravenous dose,in rats fed 5.014 ppm selenium. Radioactive selenium retention was progressively reduced in all tissues of animals fed higher concentrations of selenium.

The respiratory excretion of labelled selenium compounds was investigated in rats. A significantly higher percentage of radioactivity was excreted by respiration, and a lower percentage in the urine, in animals injected with selenite than with selenate.(32) Excretion by exhalation was essentially complete six hours after injection. Animals receiving low doses (0.08 mg selenium per kg) of selenate retained significantly more and exhaled significantly less selenium than animals receiving 1.40 mg selenium per kg. Urinary and fecal excretion percentages were unchanged. Rats injected with non-radioactive selenite two hours before receiving radioactive selenite showed no differences in exhaled selenium compared with rats receiving both simultaneously. If the cold selenite was administered 12 to 48 hours prior to the radioactive selenite, respiratory excretion of selenium was reduced to levels close to those observed when no carrier was given.(37)

Selenium oxychloride (4 and 12 mg) applied to the skin of rabbits was absorbed. A liver concentration of 2.0 to 4.3 ppm and blood level of 0.6 to 2.2 ppm resulted.(12)

3. Nonmammalian Vertebrates

Orally administered selenium-75 was used to study the absorption and excretion of selenium by hens (Gallus domestica). Selenium was absorbed at the level of the small and large intestine, and excreted by the liver, intestine, kidney, and lung as in mammals. Selenium-75 was still present in all tissues examined even 30 days after its administration.(33)

Carp contained 19 ppm selenium after a month in a 10 ppm solution of selenious acid. The mudsnail contained 90 ppm selenium after the same treatment. Duckweed contained 698 ppm.[34]

4. Plants

The accumulation of selenium by plants depends upon several factors: the species of plant, environmental conditions, age and phase of plant growth, and the nature of the selenium compounds.[12] The selenium content of plants grown in the same soil reflects the differing abilities of plants to accumulate selenium, as shown in Table 6.

Plants may be classified into three groups, based on their selenium absorption.[12] Primary selenium indicators or accumulators or "Beath's indicators" or converter plants contain large amounts of selenium (1000 to 10,000 ppm), which is mostly present in water soluble, low molecular weight, organic compounds. Secondary selenium absorbers rarely contain more than a few hundred ppm selenium on a dry weight basis, and a large percentage of the selenium is in the form of selenate. Most cultivated crop plants, grains, and grasses accumulate low concentrations of selenium (up to 30 ppm). The selenium is associated with the plant protein mainly as selenomethionine.[35]

Seasonal variations in selenium accumulation occur in indicator plants, but generally not in crop plants. Selenium concentrations are highest during periods of initial growth. Selenium accumulation depends upon the amount of selenium available to the plant in both indicator and crop plants.[12] Studies on the availability of different forms of selenium compounds showed that calcium selenate was more readily absorbed than sodium selenate, and plants absorbed more selenium as selenate than selenite. Iron (III) hydroxide selenite and sodium selenite were readily absorbed by indicator plants, but uptake by crop plants was slow. Iron (II) selenide was not absorbed by crop plants and only limited amounts were absorbed by young indicator plants. Organic selenium (extract of Astragalus racemosus) was more readily absorbed than inorganic forms as shown in Figure 2.

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Table 6

Selenium Levels in Various Species of Plants
(12)

Species	Selenium Content (ppm)
Astragalus bisulcatus	5,530
Stanleya pinnata	1,190
Atriplex nuttallii	300
Grasses	23

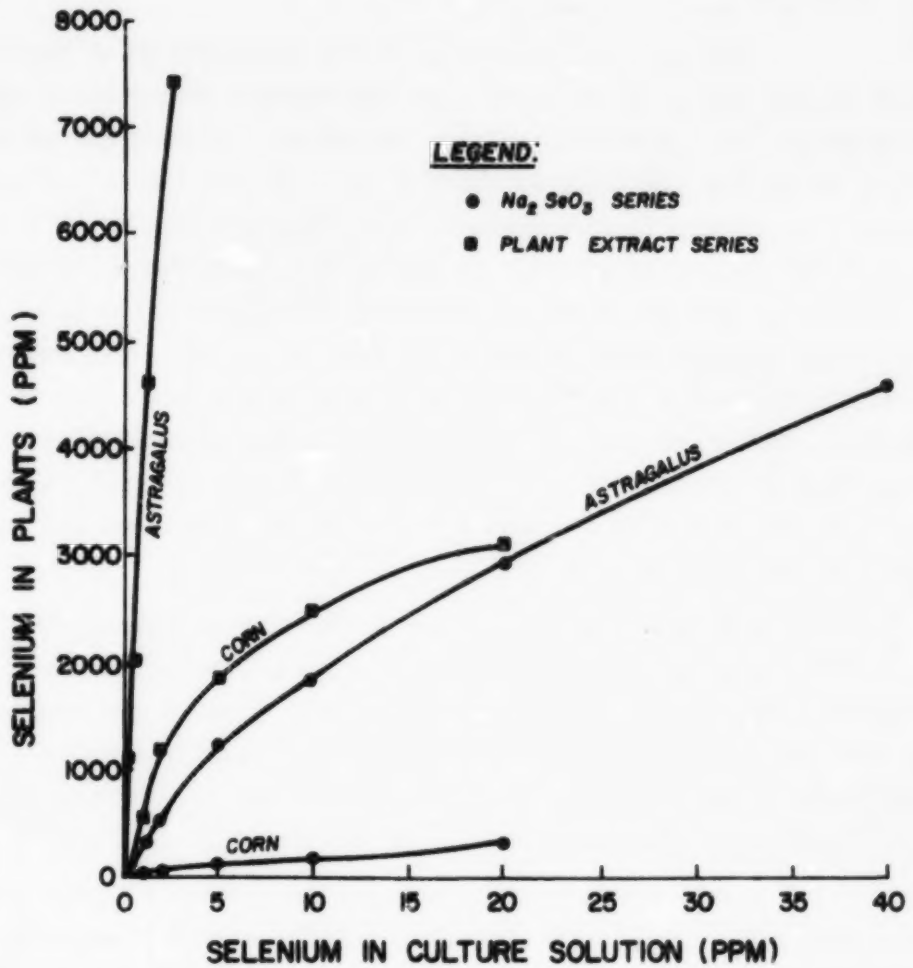

Figure 2

Comparison of the ability of corn and *A. racemosus* to accumulate selenium from selenite and from organic selenium in *Astragalus* extract

The addition of sulfate to the nutrient medium depressed selenate absorption by wheat and corn, but not the absorption of other forms of selenium.[12] Apparently sulfate and selenate compete for the same site of active uptake. Selenite is absorbed at a different site. During absorption selenite is reduced to what appears to be elemental selenium.[5] The initial step in the absorption of selenium may involve ion exchange between the plant roots and the medium. Uptake of selenium-75-labelled selenite by excised Astragalus preussii roots showed that after an initial rapid absorption of ions, an equilibrium was established between incoming and outgoing ions after five minutes. Neither an increase in time nor isotope concentration affected the percentage of selenium-75 absorption. Eighty-seven per cent of the absorbed selenium was present in the sap in the dissolved state, and the remainder was bound to the root skeleton.[12]

In some species, such as white clover, selenite was readily absorbed by the leaves and translocated to the stolons and roots.[36] Tracer studies in indicator plants have revealed that selenium absorbed by the roots is translocated to areas of active growth and metabolic activity. Selenium apparently is not stored in the plants, as aged leaves showed a loss of selenium from the mesophyll as chloride was lost.[12]

Selenium is volatilized from the leaves of intact plants and also from drying crop plants. Several volatile selenium compounds have been identified, with dimethyl diselenide comprising the largest fraction.[36]

B. Distribution

1. Human

The selenium content in human organs and tissues from subjects of various ages is shown in Table 7. In children, pancreatic selenium is extremely high compared with other tissues. In the adolescent and adults, liver and kidney tend to contain the highest levels of selenium.[25]

Table 7

Selenium Content in Human Organs and Tissues
(25)

Organ and Tissue	Child Seven Months, Extreme Pre-maturity	Child Nine Months, Intra-uterine Asphyxia	Child Nine Months, Intra-uterine Asphyxia	Child Six Months, Extreme Pre-maturity	Girl 15 Years, Food Poisoning	Man 29 Years, Skull Fracture (Trauma)	Man 64 Years, Mechanical Asphyxia	Man 75 Years, General Athero-sclerosis
Heart	0.0083	0.0040	0.0006	0.0204	0.0004	0.0072	0.0114	0.0024
Lungs	0.0020	0.0016	0.0006	0.0011	0.0024	0.0138	0.0100	0.0024
Spleen	0.0140	0.0071	0.0079	0.0046	0.0018	0.0076	0.0078	0.0014
Liver	0.0072	0.0026	0.0011	0.0019	0.0079	0.0058	0.0161	0.0072
Kidneys	0.0037	0.0028	0.0031	0.0014	0.0080	0.0271	0.0161	0.0016
Pancreas	0.0260	0.0240	0.0261	0.0260	0.0032	0.0026	0.0100	0.0021
Muscles	0.0021	0.0021	0.0024	0.0016	0.0014	0.0028	0.0226	0.0020
Skin	0.0036	0.0021	0.0025	0.0028	0.0012	0.0028	0.0124	0.0032
Bone	0.0105	0.0090	0.0064	0.0984	0.0021	0.0031	0.0243	---

2. Other Mammals

The highest concentrations of selenium in rats fed experimental diets for 70 days occurred in the kidneys in all the groups. The liver contained the next highest concentration, followed by the spleen and blood, the skin, hair, and muscles.[30] In lambs fed varying selenium intakes, the highest retention of oral or intravenous selenium-75 was in the pelt and whole blood, followed by the kidney, liver, and lung. The percentage of the radioactive dose retained was much greater in all tissues of animals fed low selenium diets. Adipose tissue retained much less selenium-75 than muscle or bone.[31]

The distribution of selenium-75 injected as selenate in the blood of rats varied with the amount of selenium injected. The fraction of injected dose per ml appearing in the red blood cells increased almost tenfold as the selenium dose was increased from 0.08 to 1.4 mg selenium per kg. The total serum lipoprotein selenium content more than doubled as the selenium dosage was increased, and most of this effect could be attributed to a tenfold increase in the selenium content of the ether-acetone extractable lipid fraction.[32] The alpha-2-globulin fraction of serum proteins contained the highest amounts of injected selenium.[37]

In animals receiving very low dietary levels of selenium, the highest selenium concentrations usually occur in the kidney, followed by the liver and muscle. The selenium level in the liver rises more rapidly than in the kidney as the level of dietary selenium is increased.[28]

Selenium-75 appears in the milk of dogs injected with radioactive selenite 278 days previously. Ewes fed a high selenium diet for six months, followed by a low selenium diet may transmit protective levels of selenium to their lambs born nearly one year after the ewes were placed on the low selenium diet.[28]

The pups of female rats maintained on selenium-supplemented diets for 70 days before mating contained more than four times as much selenium on day one as the pups of dams continuously maintained on low selenium diets. The birth weights of the pups were not significantly different, but at 55 days of age, weights of pups of selenium-supplemented dams were double the weights of pups of low selenium dams. The selenium levels of all groups of pups were not significantly different on day 55 in the surviving pups. The kidneys had the highest selenium levels. Mortality data indicate that pups of dams fed

0.078 to 0.093 ppm selenium supplements prior to breeding had a higher survival value than pups of dams fed low (0.024 ppm) or higher (0.120 to 0.146 ppm) selenium for the same period.[30]

The effects of dietary intake of selenium on the distribution of selenium in the organs of yearling ewes are shown in Table 8.[35] High selenium intake resulted in high selenium in the liver; high levels also occurred in the kidneys. Selenium apparently cleared from the animal tissues to some extent once the selenium supplements were withheld, but organ selenium was still higher than for animals fed basal diet. The liver and kidneys can accumulate quite high levels of selenium if dietary selenium is high. No significant differences in muscle, heart, or liver selenium levels were observed between swine receiving dietary selenium supplements of 0.1 ppm or 1.0 ppm.[38] See Table 9. The apparent differences in tissue accumulation of selenium between the ruminant sheep and the monogastric swine may be due to excretion differences, basal diet selenium levels, absorption differences, or other species differences.

3. Nonmammalian Vertebrates

Endogenous selenium distributed in the tissues of three avian species receiving chicken breeder pellets containing 0.27 ppm selenium was studied.[39] Leghorn chickens is shown in Table 10 for two ages. The pineal concentration was extraordinarily higher than in the other tissues, especially in the 24-week old males. The liver and kidney accumulated the largest concentrations of injected selenium-75, and the older birds accumulated twice as much as did the young birds.

The tissue distribution of endogenous selenium is shown in Table 11 for female chickens, turkeys, and Japanese quail (Coturnix coturnix japonica) at the laying stage. The pineal selenium in all species is much less than that observed in the male chickens. The pituitary and kidney of all species contained high selenium concentrations. The pancreas of Coturnix also contained quite high selenium. The fecal and crop selenium concentrations were about the same. Egg yolks had a much higher selenium content than the whites or shells.

Table 8.

Effects of Selenium Supplements on Tissue Levels of Selenium in Yearling Ewes (35)

Diet	Selenium Content (ppm) Kidney	Liver	Muscle	Heart	Fat
Basal (low Se)	0.5	0.75	0	—	0
8 mg Se per day-44 days 64 days-basal diet	1.57	1.85	0	0.57	0
10 mg Se daily-64 days	6.87	9.10	0.8	1.90	0
20 mg Se daily-44 days 61 days-basal diet	1.47	1.33	0	0.99	0
17 mg Se daily-116 days	3.95	29.21	0.8	1.75	0

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Table 9.

Effects of Selenium Supplementation on Tissue Selenium Levels in Swine (38)

	Selenium Level (ppm)	
Tissue	Basal diet + 0.1 ppm Se	Basal diet + 1.0 ppm Se
muscle	0.16	0.15
heart	0.21	0.17
liver	0.56	0.52

Table 10.

Endogenous Levels of Selenium in the Tissues of Male White Leghorn Chickens (39)

Tissue	Selenium Content (ppm) 24-Week Old	Year-Old
Blood	0.329	0.634
Pineal	18.85	4.04
Liver	0.737	0.499
Kidney	0.934	0.681
Cerebellum	0.278	0.277

Table 11.

Endogenous Selenium Levels in the Tissues of the Mature Females of Three Avian Species (39)

	Selenium Content (ppm)		
Part	Chicken	Turkey	Coturnix
Blood	0.182	0.166	0.459
Pineal	1.27	0.364	0.380
Pituitary	1.16	0.982	1.11
Eye	0.095	0.072	0.120
Kidney	0.649	1.077	1.50
Spleen	0.539	0.705	0.950
Liver	0.475	0.800	0.887
Pancreas	0.364	0.499	1.259
Magnum	0.295	0.182	0.348
Cerebrum	0.230	0.230	0.356
Diencephalon	0.222	0.190	0.309
Cerebellum	0.214	0.301	0.372
Ovary	0.182	0.364	0.491
Pectoral	0.174	0.190	0.301
Feces	0.079	0.127	0.198
Crop contents	0.071	0.127	—
Egg yolk	0.531	—	0.626
Egg albumin	0.103	—	0.135
Egg shell	0.067	—	0.143
Ration	0.271	0.337	0.337

In 14- and 20-week old turkeys, the addition of 0.1 or 0.2 ppm selenium to the diets produced no significant differences in the selenium levels in the breast muscle, liver muscle, liver, or blood.[38] In broiler chickens, the addition of 0.1 to 0.4 ppm selenium to a basal diet containing 0.07 ppm selenium resulted in some effects on tissue selenium levels as shown in Table 12. Dietary levels of 0.2 ppm selenium caused increases in both muscle and kidney selenium content. Skin selenium was also somewhat increased by the supplementation. Dietary selenium at 0.1 ppm apparently increased liver selenium content, but higher levels reduced it. In another study, no changes in thigh or breast muscle selenium in chickens were produced by the addition of 2.0 ppm selenium to the diet. Liver, kidney, and heart selenium were increased by this supplementation.[38] Dietary supplementation of organic selenium more effectively increases tissue concentrations of selenium than inorganic supplements.[26]

The addition of 0.1 ppm of sodium selenite to the diet of laying hens increased laying capacity by 10 per cent and increased the selenium content of liver, ovary, heart, pectoral muscle, blood cells, and kidney.[40] Selenium accumulation was greater in the kidneys of nonlayers and less distinct in the kidneys of layers, presumably due to the increased selenium deposition in the eggs of layers.[47] The selenium content in the eggs of hens fed 0.1 ppm selenium supplements was higher than in nonsupplemented hens' eggs as shown in Table 13.

The incubating eggs of hens receiving 36 micrograms of selenium daily in their diets showed steadily increasing amounts of selenium in their yolk sac and in the liver, blood, and proteins, but not in the heart, of developing embryos.[43]

Studies with radioactive compounds revealed that injected selenite, but not selenate, becomes bound to the serum proteins of chicks. At low levels, binding is predominantly to the alpha and gamma globulins, but at higher concentrations, albumin binding also takes place. Treatment of the serum proteins from chicks injected with both radioactive selenate and selenite with dilute alkali released 85 per cent of the bound selenium-75 from the protein of which more than half was selenite.[44]

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Table 12.

Effects of Selenium Supplementation on Tissue Selenium Levels in Broiler Chickens (38)

	Selenium Content (ppm)			
Tissue	Basal	Basal + 0.1 ppm Se	Basal + 0.2 ppm Se	Basal + 0.4 ppm Se
muscle	0.061	0.071	0.103	0.114
liver	0.25	0.48	0.34	0.13
kidney	0.39	0.34	0.80	0.56
skin	0.09	0.13	0.16	0.13

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Table 13.

Effect of Selenium Supplements on Selenium Content of Hens' Eggs (42)

Diet	Selenium Content (micrograms) Albumin	Yolk
Basal	1.9	3.14
Basal + 0.1 ppm Se	2.34	3.64

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4. Plants

The distribution of selenium in plants is quite different for indicator plants compared with crop plants. Table 14 shows that in most indicator species, higher selenium concentrations occur in the tops of the plants then in the roots; for crop plants, the opposite is true; the roots have a higher selenium content than the leaves and stems as shown in Table 15. The distribution of selenium in the parts of mature corn grown in sand culture with various concentrations of selenium added as either selenite or organic selenium is shown in Table 16. Supplemental organic selenium (2.5 and 5.0 ppm) produces much higher selenium accumulation in all parts of the corn than the selenite.

C. Growth and Nutrition

1. Humans

Selenium has been demonstrated to be a definite nutritional requirement in animals. Some preliminary findings suggest the necessity of selenium in human nutrition as well.

In children treated for kwashiorkor in Jamaica, satisfactory weight gain responses were not achieved until the diets were supplemented daily with 0.25 mg of selenium as γ, γ-diseleno-di-n-valeric acid.[45] Similar effects were observed in infants suffering from malnutrition in Jordan.[46] Oral administration of 0.30 mg of selenium as sodium selenite daily in milk stimulated weight gains. Selenium is reduced in the whole blood, plasma, formed elements and plasma proteins of victims of kwashiorkor compared with healthy adults or children recovered from kwashiorkor.[46]

2. Mammals

Selenium deficiency diseases are not uncommon in ruminants and nonruminants, mammals, and avians. In rodents, hepatic necrosis accompanied by renal necrosis, pancreatic dystrophy, and skeletal and heart muscle degeneration are the symptoms of severe selenium deficiency. Mitochondrial and cytoplasmic changes in the liver can be detected by electron microscopy before gross necrosis is evident. Death may occur in four to six weeks unless selenium supplements are supplied. Vitamin E is also effective in alleviating symptoms.[35]

Table 14.

Distribution of Selenium in Tops and Roots of Selenium Accumulators (12)

Plant	Selenium (ppm)		Ratio
	Tops	Roots	Tops/roots
Astragalus crotalariae	2,000	45	44
A. beathii	1,963	66	30
A. pattersoni var. praelongus	583	26	22
A. argillosus	385	27	14
A. bisulcatus	1,180	130	9
A. preussii	313	40	8
A. beathii (greenhouse, selenate)	524	213	2.5
A. pattersoni	730	296	2.5
A. racemosus (greenhouse, selenite)	1,256	659	1.8
Haplopappus fremontii	4,800	4,800	1.0
Stanleya pinnata	125	429	0.3

Table 15.
Distribution of ^{75}Se Between Roots and Tops (36)

Species	^{75}Se Concentration (1000's of CPM/Gm. Dry Wt.)		Concentration Ratio
	Roots	Tops	Roots / Tops
Spinach	2578	110	23.4
Perennial ryegrass	1802	115	15.6
Tomato	1753	138	12.7
Mustard	1760	163	10.8
Barley	1886	176	10.7
Sunflower	2507	262	9.6
Wheat	1774	195	9.1
Annual ryegrass	2194	256	8.6
Alfalfa	1561	207	7.5
Subterranean clover	3979	538	7.4
Phalaris tuberosa	1635	229	7.1
Onion	650	246	2.6

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Table 16.

Selenium Content of Mature Corn Grown in Sand Cultures (12)

Selenium added (ppm)	Selenium (ppm)							
	Roots		Stems		Leaves		Grain	
	A[a]	B[b]	A[a]	B[b]	A[a]	B[b]	A[a]	B[b]
0.5	135	121	60	78	58	86	77	55
1.0	155	218	105	138	76	153	107	99
2.5	209	817	147	393	141	359	209	325
5.0	321	1,820	182	1,228	134	890	308	694

Selenium added to culture solutions:

[a]A: sodium selenite

[b]B: selenium as aqueous extract of A. bisulcatus

In swine, hepatosis diaetetica is characterized by liver necrosis, degeneration of the muscles and heart, massive transudations, and ceroid pigments in the adipose tissue. Nutritional muscular dystrophy is accompanied by increased levels of serum glutamate oxaloacetate transaminase. Death results in 22 to 45 days. Diets containing 0.5 ppm selenium as sodium selenite or 100 ppm vitamin E, or both, reduced the incidence of mortality. Cystine was ineffective.(35)

In horses in New Zealand, neonatal myopathy could be relieved by injections of sodium selenate at birth and at 10 days of age. The syndrome is characterized by watery, chalky white, degenerating musculature, loss of hair, and abnormally thick, firm layers of yellow-brown fat.(35)

3. Other Mammals

White muscle disease (WMD) occurs in lambs and calves whose dams have been fed diets containing less than 0.02 ppm selenium in the dry matter. The symptoms are severe bleaching and calcification of skeletal and heart muscles, resulting in a loss of normal motor function and deformed limbs. High levels of serum glutamate oxaloacetate transaminase and creatinuria accompany the syndrome.(35)

WMD can be prevented by feeding ewes 0.1 ppm selenium in the diet. One hundred mg of sodium selenite per 30 kg body weight administered orally to cows prevents WMD in calves. Lambs and calves can also be treated prophylactically with selenium compounds. Vitamin E alone was ineffective.(35)

Selenium is required for normal reproduction and growth of offspring. In New Zealand, the percentage of lambing in certain areas was increased from 62 per cent to 94 per cent by the oral dosing of ewes with 5 mg of selenium as sodium selenite prior to breeding.(35)

Selenium-depleted rats were obtained by feeding a purified low selenium diet containing vitamin E to weanling rats.(47) After twenty weeks, selenium levels in the blood, liver, and skeletal muscles had been reduced from 0.32, 0.60 and 0.23 ppm to 0.05, 0.04 and 0.02 ppm, respectively. Seventy per cent of these declines occurred within the first six weeks. Improved growth and increases in liver and blood selenium were observed 20 weeks after beginning a dietary supplement of 0.5 ppm selenium as seleno-methionine. Females

maintained on the low selenium diet reproduced normally through three litters, but the offspring had reduced growth and little hair compared with offspring fed a selenium-supplemented diet or born to dams on a supplemented diet.

4. Birds

Exudative diasthesis in chicks apparently develops due to a lack of ability to absorb sufficient vitamin E.[35] The symptoms include edema, hemorrhage, anemia, degeneration of hemoglobin, and decrease in albumin. The abdominal wall takes on a greenish-blue appearance. Symptoms appear two to three weeks after chicks are placed on a diet deficient in both selenium and vitamin E. The absence of selenium produces fibrotic pancreatic degeneration with an accompanying decrease in lipase. As a result, lipid digestion is severely impaired. Undigested lipids cannot be absorbed and the absorption of fat-soluble vitamin E is extremely reduced. Relief can be obtained by supplementing the diet with large amounts of vitamin E, so that enough is absorbed, or with selenium to restore normal pancreatic function. The amount of selenium necessary is directly related to the amount of vitamin E provided in the diet. Only 0.01 ppm selenium as sodium selenite is needed if 100 International Units of vitamin E per kg are present. With only 10 to 15 units, 0.02 to 0.04 ppm of dietary selenium is necessary for relief of the symptoms.

In turkeys, selenium deficiency is manifested by severe degeneration of the gizzard musculature. Exudative diasthesis may be present, but is not as severe as in chicks. Anemia and generalized myopathy are accompanying symptoms.[35]

Poor feathering and wasting of the entire body are symptoms of selenium deficiency in Japanese quail. Extremely frail young are produced by females fed very low selenium diets (0.002 to 0.005 ppm). If the young are maintained on the same low-selenium diets, death occurs by day 25. Vitamin E supplementation does not relieve the condition.[35]

5. Plants

In indicator plants, growth effects of selenium are not always the same.[36] The growth of Astragalus racemosus was greatly stimulated by the presence of selenite or selenate in the medium. The growth of other

species in the same genus was actually inhibited by the presence of high selenium, although selenium uptake was pronounced. The growth of Astragalus racemosus was reduced when the selenium source was Astragalus extract. Selenium accumulation occurred at a higher level than with inorganic selenium, however.

In the crop plants alfalfa and white clover, no beneficial effect of selenium on plant growth was observed at even very low concentrations of selenium (0.099 ppb). Apparently these crop plants have no nutritional requirement for selenium.[36]

6. Microorganisms

The micronutrient requirement of selenium for microorganisms has not been investigated. However, the induction of formic acid dehydrogenase in Escherichia coli in a highly purified medium requires the addition of 10^{-8} molar selenite.[48]

Compounds of selenium have different affects on the growth of microorganisms as shown in Table 17.

Table 17.

Effect of Selenium Compounds on Growth of Microorganisms

Organism	Effect on Growth	Selenium Compound
Chlorella vulgaris	Increase in cell size	selenomethionine
Lactobacillus casei	Inhibition	selenopurine, seleno-quanine
Saccharomyces cerevisiae	Inhibition	selenate
Escherichia coli	Inhibition	selenate
Yeast	Stimulate cell division	selenite
Paramecium caudatum	Inhibition	selenite
Neurospora crassa	Inhibition	selenate
Aspergillus niger	Inhibition	selenate

D. Biochemistry

1. Animals

a. Fate of inorganic selenium compounds

Selenite and selenate are rapidly incorporated into similar protein fractions of the blood and other organs soon after administration to animals. The initial plasma binding is to albumin, but the bulk of the selenium remaining in the blood is bound to the alpha globulin fraction, from which it is slowly excreted. Inorganic selenium detaches from albumin and enters various other cells of the body where it can catalyze the oxidation of sulfhydryl groups. The body attempts to detoxify selenite in the liver by reductive methylation to the trimethylselenonium ion, which can be excreted by the kidney, or dimethyl selenide which can be excreted by the lungs.(49)

Originally, inorganic selenium compounds were thought to undergo conversion to selenoamino acids, followed by their incorporation into proteins. This hypothesis is contradicted by a number of experimental findings: the rapidity with which selenium appears in the protein; the ease with which it is removed by dialysis with dilute alkali or treatment with sulfhydryl reagents or urea; and the concentration of selenium in a limited number of protein fractions as demonstrated by gel filtration, electrophoresis, and autoradiography. Apparently selenite and selenate bind to the sulfhydryl bridges of proteins directly or as other inorganic ions such as selenotrisulfide.(50)

In vitro studies indicate that mammalian tissues are able to reduce selenite to elemental selenium. Fresh tissue homogenates reduced both selenate and selenite to elemental selenium. Selenite was reduced more readily. The amount of reduction varied from tissue to tissue.(12)

b. Fate of organoselenium compounds

Selenomethionine is actively transported across cell membranes by the same mechanism as methionine, but selenocystine is apparently not accumulated by the cystine mechanism.(12)

Selenium amino acids apparently undergo the same metabolic reactions as their sulfur analogs.[49] Selenomethionine and selenocystine are incorporated into proteins, and converted into selenium analogs of sulfur metabolites. Thus selenoglutathione, selenotaurine, selenocysteic acid and selenocystathionine are produced from selenomethionine and selenocystine in the rat and chick.

Radioactive seleno amino acid analogs are often used in studies of protein synthesis.

c. Antioxidant function

Selenium compounds apparently have the capacity to act as biochemical redox agents and to function as biological antioxidants.[51] Highly reactive free radicals and peroxides may form in the body as a result of antioxidant deficiency, radiation damage, the aging process and oxygen toxicity. Free radical products of lipid peroxidation, for example, in the absence of sufficient antioxidants such as vitamin E and ascorbic acid, can cause significant alterations in the structural and functional components of the cell by initiating chain reactions. Comparative studies on *in vitro* peroxidation inhibition by proteins from control and selenium-supplemented animals indicated that selenoproteins are the superior antioxidants, inhibiting peroxidation 20 to 500 per cent better than control proteins and 50 to 500 times more efficiently than vitamin E.

Selenium compounds were more efficient peroxide decomposers. Selenocystine consumed up to three moles of peroxide oxygen per mole compared with methionine, which consumed one mole of peroxide per mole of amino acid. Free radicals in tissues produced by gamma irradiation with cobalt-60 damage proteins and amino acids:[51] Selenomethionine was a more effective protector of both labile and stable amino acids against radiation damage than methionine. Presumably functioning as a free radical scavenger, selenomethionine also had 5.2 and 3.3 times the protective activity as methionine, and selenocystine had 3.2 and 1.9 times the protective activity of cystine for alcohol dehydrogenase and ribonuclease, respectively. Selenocystine was also a more effective protector of enzymes against loss of activity by peroxidation.

Selenocystine promotes the oxidation of low molecular weight thiols such as reduced glutathione and cysteine while protecting sensitive sulfhydryl groups on enzymes.(51)

Recently selenium was found to be a required cofactor for glutathione peroxidase in rats.(13) Glutathione peroxidase protects hemoglobin from peroxidation by destroying hydrogen peroxide by reacting it with reduced glutathione. Selenium is apparently an integral part of the enzyme required for its activity. The precise role of the selenium in the mechanism of the reaction has not as yet been elucidated. Purified ovine glutathione peroxidase contains at least two atoms of selenium per molecule of enzyme.

2. Plants

Of the total selenium in the tops of non-accumulator plants, 70 to 90 per cent is usually found in the insoluble fraction; most of this can be solubilized by proteolytic enzymes. In contrast, in indicator plants, soluble selenium compounds are present in the largest amounts.(28)

Selenomethionine in protein is the dominant form of selenium in non-accumulator plants. Selenomethylselenocysteine in the soluble fraction was the dominant form of selenium in some indicator species.

The reduction of selenate to selenite may be the rate limiting step in the utilization of selenium by plants. In the roots, selenite is converted to elemental selenium. Selenocysteine is formed from selenite by an unknown mechanism. Selenocysteine is converted to selenoglutathione, selenomethylselenocysteine in indicator plants, selenocystathione in non-accumulator plants, and incorporated into protein in non-accumulators, and to a small extent, in indicators. In non-accumulators, selenocystathione is converted to selenomethionine, which is mostly incorporated into protein, but may be converted to selenomethylselenomethionine.(28)

The forms of selenium present in a number of native plant species are summarized in Table 18. Inorganic selenium was present as selenate, but no selenite was found. Plants containing the highest percentages of inorganic selenium are usually the most toxic to livestock.[12]

Presumably, organoselenium compounds can be absorbed directly by the plant, and, therefore, can be more quickly metabolized than selenate, which must first be converted to selenite.

3. Inhibition of Metabolic Reactions by Selenium Compounds

Selenium compounds apparently inhibit enzymatic reactions in two ways:[25] inorganic selenite and selenate interfere with sulfhydryl groups on the enzyme surface;[12] organic and inorganic selenium compounds compete with their sulfur analogs. Some reactions inhibited by selenium compounds appear in Table 19.

4. Miscellaneous Actions

Selenium may replace sulfur in some reactions and stimulate others. Table 20 summarizes some of these effects.

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Table 18.

Organic Selenium and Selenate in Native Plants (12)

Plants (arranged in order of decreasing % organic Se)	Total Se in plant (ppm)	Forms of Se		Water-soluble Se (%)
		Organic (%)	Selenate (%)	
Castilleja chromosa	1,812	95	5	57
Agropyron smithii (Wyo.)	98	91	9	63
Agropyron smithii (Wyo.)	41	85	15	51
Oryzopsis hymenoides (Utah)	93	78	22	57
Comandra pallida	140	74	26	41
Haplopappus fremontii	680	70	30	79
Agropyron smithii (Utah)	202	51	49	78
Gutierrezia sarothrae	120	50	50	67
Haplopappus engelmanii	101	47	53	74
Castilleja angustifolia	250	46	54	84
Grindelia squarrosa	102	45	55	78
Haplopappus fremontii subsp. wardii	932	42	58	67
Gutierrezia diversifolia	723	37	63	71
Aster commutatis	325	25	75	92
Machaeranthera venusta	3,486	24	76	89
Machaeranthera glabriuscula	1,431	23	77	91
Aster occidentalis	284	23	77	99
Atriplex canescens	477	19	81	84
Hymenoxys floribunda	575	16	84	94
Machaeranthera ramosa	1,345	15	85	98
Atriplex confertifolia	1,734	10	90	98
Aster caerulescens	560	9	91	98
Atriplex nuttallii	502	8	92	95

Table 19.

Metabolic Reactions Inhibited by Selenium Compounds (12,48)

Inhibited Reaction or Enzyme	Reaction Site	Selenium Compound
Transmethylation	Liver homogenate	selenite, selenate
Glycolysis (nitrogen atmosphere)	Rat liver homogenate	selenite
Succinic dehydrogenase	Muscle powder	selenite
DNA depolymerization by DNAse	Pancreas	selenite
Respiration	Tissue slices	selenite
Succinoxidase	---	selenite
Purified D-amino acid oxidase	---	selenite (0.001M)
Purified B-amylase	---	selenite (0.001M)
Purified urease	---	selenite (0.001M)
Respiration	Chick embryonic cartilage	selenate
Chondroitin sulfate synthesis	Chick embryonic cartilage	selenate
Cell division	Chlorella vulgaris	selenomethionine
Fermentation	Yeast	selenate, selenite, selenide
Glycolysis	Yeast	selenite
Respiration	Yeast	selenite
Oxygen uptake	Yeast	selenite
Triose phosphate dehydrogenase	Yeast	selenite
Isocitrate dehydrogenase	Yeast	selenite
B-galactosidase synthesis	Methionine-requiring Escherichia coli	selenomethionine
Sulfate reduction	Desulfovibrio desulfuricans	selenate
Growth	Mouse leukemia cells	selenopurine
Oxygen consumption	Paramecium caudatum	selenite
Chlorophyll synthesis	Crop plants	selenate

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Table 20.

Noninhibitory Metabolic Actions of Selenium
(12,48,51)

Action	Organism	Selenium Compound
Fumarase activation	Pig (heart)	selenate (0.025M)
Methylation substrate	Fungi	selenite, selenate
Methyl donor	Yeast	selenomethionine
Formate dehydrogenase induction	Escherichia coli Azotobacter aerogenes	selenite
Incorporation into protein	E. coli Candida albicans	selenomethionine, selenite
ATP sulfhydrylase substrate	Yeast	selenate
Glutathione peroxidase component	Rat	(?)

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IX ENVIRONMENTAL EFFECTS

A. Environmental Content, Transport, Contamination

The natural content of selenium in the soil and rocks is the largest source of selenium in most environments.[12] Both the presence and absence of selenium can provide biological hazards.

Soil selenium enters the food chain by plant accumulation. In seleniferous soils, certain species of plants prevail which easily accumulate selenium levels toxic to livestock. In fact, the presence of certain plant species is steadfast evidence of a seleniferous zone. Crop plants grown on such soils, if they themselves survive, may also contain selenium levels poisonous to livestock. The distribution of some seleniferous areas in the western United States is reflected in Figure 3, which shows the distribution of seleniferous vegetation in that area.[12]

In areas where soil selenium is extremely low, forage plants contain very little selenium. Livestock ingesting food raised in these areas produce offspring which exhibit severe deficiency diseases. The symptoms depend upon the species. Figure 4 illustrates the lack of overlap between areas in the United States where white muscle disease, a selenium deficiency syndrome, occurs, and areas supporting seleniferous vegetation.[12] The map in Figure 5 shows the distribution of the selenium content of forages in the Pacific Northwest.[13] The coincidence of high WMD areas shown in Figure 4 with forages containing very low selenium as shown in Figure 5 is evident. Areas with adequate forage selenium show no incidence of WMD.

Waters in seleniferous areas may contain toxic selenium levels. Drainage from irrigation and soil leaching are principal sources of selenium in waters. In Nebraska, water from 42 per cent of all wells sampled and from one quarter of all surface water locations contained selenium in greater concentrations than the U.S. Public Health Service standard (10 µg/l).[14] The amount of selenium which is carried to sea water is great enough to cause serious poisoning of the ocean had there been no mechanism for the removal of selenium from sea water.[12] Selenium is removed from aqueous solution by

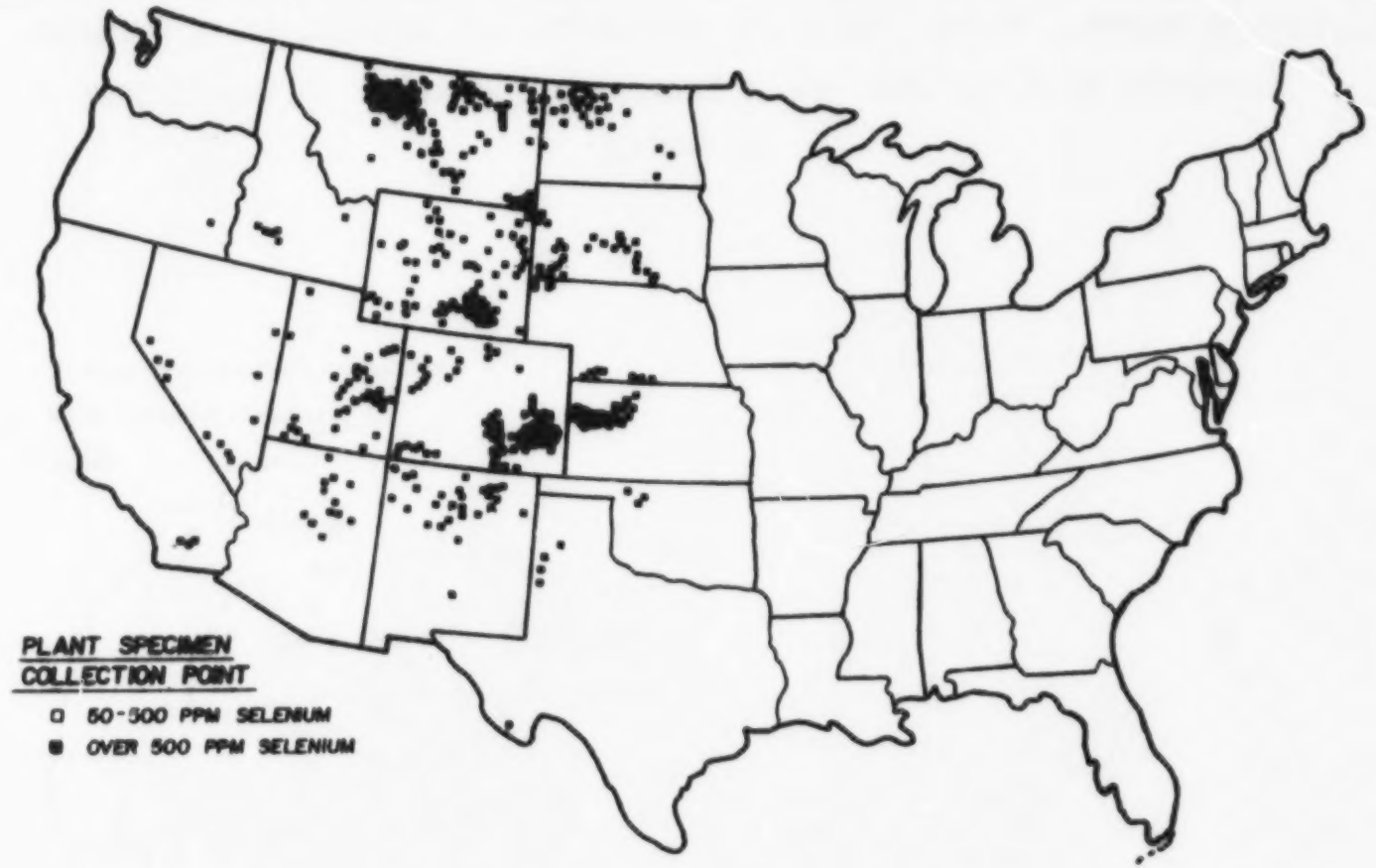


Figure 3

Distribution of seleniferous vegetation in the western United States

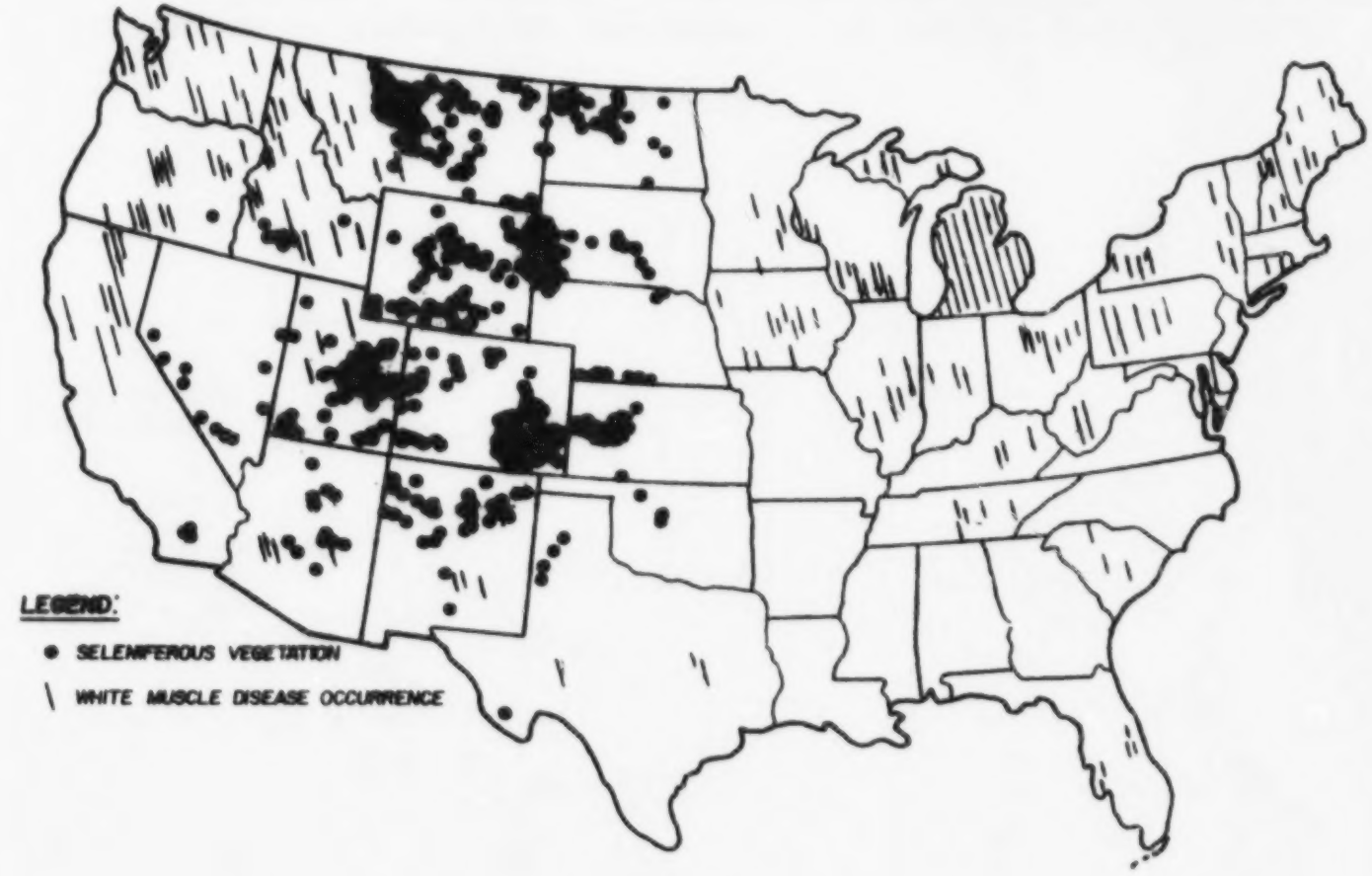
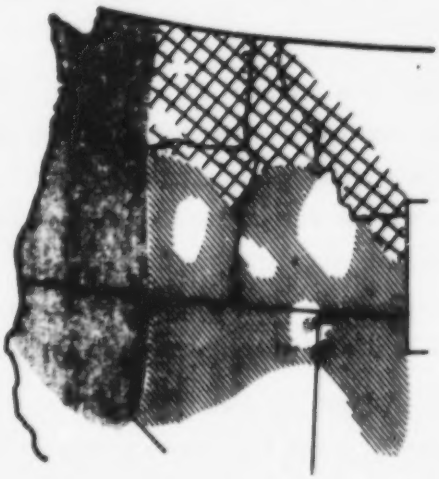


Figure 4

Distribution of vegetation containing more than 50 ppm selenium, in relation to distribution of occurrence of white muscle disease in livestock



VERY LOW Se - LESS THAN 0.10 PPM IN MORE THAN 95% OF SAMPLES AND LESS THAN 0.05 PPM IN MORE THAN 80% OF SAMPLES

LOW Se - LESS THAN 0.10 PPM IN MORE THAN 80% OF SAMPLES AND LESS THAN 0.05 PPM IN MORE THAN 60% OF SAMPLES

VARIABLE Se - LESS THAN 0.10 PPM IN 50% OF SAMPLES

ADEQUATE Se - MORE THAN 0.10 PPM IN 90% OR MORE OF SAMPLES

Figure 5

Selenium content of forages in the Pacific Northwest

adsorption on precipitated hydroxides of iron and manganese, organic matter and iron sulfides.[12] Nearly all sea floor samples analyzed contain selenium. Values reported range from 0.03 to 2.0 ppm selenium.[12] (See Table 21)

Coal contains variable amounts of selenium.[15] Table 22 shows some reported values. The burning of fuels containing selenium may represent a significant source of the element in the environment. The selenium content of atmospheric dust collected on air conditioning filters in various industrial areas in the United States are presented in Table 23.[15] The range of selenium contents found in some foods which are ingested by humans are presented in Table 24. Animal products contain considerable amounts of selenium.[12]

In one study, an attempt is made to identify the source of atmospheric selenium pollutions by comparing the selenium:sulfur ratio of various fuels with that ratio in air particulates and in top soils. The data are presented in Table 25. Selenium:sulfur ratios for petroleum samples are of the same order of magnitude as the air samples collected.[16]

Most fresh vegetables and fruits for human consumption contain quite low amounts of selenium as shown in Table 26. Starchy vegetable products contain much higher selenium concentrations as shown in Table 27. Table 28 presents the selenium content of meats and seafoods. Kidneys have notably high selenium content. Seafood has generally higher selenium content than meat. The selenium contents of dairy products are, in general, lower than for meats, and higher than for fruits and vegetables. (Table 29) Table 30 shows the variable selenium content of eggs and some sweeteners. The content of baby foods appears in Table 31. The lower selenium content in baby foods compared with fresh may be due to volatilization of selenium during the cooking. All food samples analyzed were purchased in Beltsville, Maryland, northeast of Washington, D.C.[17]

Table 21.

Selenium in Sea Water
(15)

Area	No. of Samples	Selenium (Mcg./l) Range	Average
Caribbean	4	0.095-0.14	0.11
Western North Atlantic	8	0.069-0.13	0.096
Eastern North Atlantic	7	0.076-0.11	0.088
Western South Atlantic	2	0.070-0.080	0.075
Eastern Pacific	6	0.061-0.12	0.087
Antarctic	1	0.052	0.052
Long Island Sound	8	0.10 -0.13	0.11

Table 22.

Selenium in Coal
(15)

Area	No. of Samples	Selenium (ppm) Range	Average
		Lignite	
Western Wyoming	3	1.96-7.38	3.88
Montezuma County, Colorado	2	2.0 -2.4	2.2
		Coal	
Morley, Alberta, Canada	1	---	2.0
Sandoval County, New Mexico	1	---	0.1
Kladno, Czechoslovakia	-	Present	---
Liege, Belgium	-	Present	---

Table 23.

Selenium Content of Atmospheric Dust From Air-Conditioning Filters
(15)

Type of Building Where Sample Was Collected	Location	Selenium (ppm)
Industrial	Los Angeles, California	0.8
Dry goods store	San Francisco, California	0.05
Industrial	San Leandro, California	0.6
Residence	Grand Forks, North Dakota	6.0
Residence	Houston, Texas	3.0
Residence	University City, suburb of St Louis, Missouri	2.5
Office Building	St. Louis, Missouri	10.0
Unknown	Chicago, Illinois	2.5
Residence	Shaker Heights, suburb of Cleveland, Ohio	2.5
Unknown	Philadelphia, Pennsylvania	1.5
Office Building	Washington, D.C.	0.5

Table 24.

The Selenium Content of Water, Milk, Eggs, Meat, and Bread
(12)

Material	Total Number of Samples	Number of samples showing:			Selenium (ppm)	
		No Selenium	Traces	Positive	Minimum*	Maximum
Water	44	20	14	10	0.05	0.33
Milk	50	0	6	44	0.16	1.27
Eggs	32	0	0	32	0.25	9.14
Meats	6	0	0	6	1.17	8.00
Bread	11	0	5	6	0.25	1.00

*Positive samples only

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Table 25.

Selenium:Sulfur Ratio of Some Possible Sources of Air Pollution and Some Environmental Samples (16)

Sample	Selenium (μg/g)	Sulfur (mg/g)	Se/S Ratio ($x10^4$)
Raw petroleum	0.92	18.4	0.42
Heavy petroleum A	1.10	10.9	1.03
Heavy petroleum B	0.75	16.9	0.55
Heavy petroleum C	1.12	19.3	0.58
Rubber tires	1.33	13.0	1.01
Coal	1.18	2.44	4.85
Soot (mechanical collection)	4.30	138.00	0.31
Soot (electrical collection)	0.50	30.00	0.17
Soil	1.14	1.76	6.84
Soil extract	0.50	0.11	0.48

Table 26.

Selenium Content of Vegetables and Fruits
(17)

Products	µg Se/gram	
Vegetables		
Carrots		
Fresh	0.022,	0.022
Canned	0.013,	0.013
Cabbage, fresh	0.022,	0.023
Cauliflower, fresh	0.006,	00.007
Corn		
Fresh	0.004,	0.004
Canned	0.003	0.005
Garlic, fresh	0.276,	0.222
Green pepper, fresh	0.006,	0.008
Green beans		
Fresh	0.006,	0.006
Canned	0.009,	0.009
Lettuce, fresh	0.009,	0.007
Mushroom		
Fresh	0.122,	0.141
Canned	0.109,	0.100
Onion, white, fresh	0.015,	0.015
Potatoes		
Sweet, fresh	0.007,	0.006
White, fresh	0.003,	0.006
White, canned	0.007,	0.011
Radish, fresh	0.042,	0.036
Tomatoes		
Fresh	0.005,	0.005
Canned	0.010,	0.009
Turnips, fresh	0.006,	0.008
Mean excluding mushroom and garlic	0.010	
Fruits		
Apple, fresh, peeled	0.003,	0.006
Applesauce, canned	0.002,	0.002
Banana, fresh, peeled	0.010,	0.009
Orange, fresh, peeled	0.014,	0.012
Peach		
Fresh, peeled	0.004,	0.004
Canned	0.004,	0.002
Pear		
Fresh, peeled	0.006,	0.006
Canned	<0.002,	<0.002
Pineapple		
Fresh	0.006,	0.005
Canned	0.008,	0.012
Mean	0.006	

Table 27.

Selenium Content of Grains and Cereal Products
(17)

Product	µg Se/gram	
Barley cereal	0.643,	0.676
Bread		
White	0.280,	0.274
Whole wheat	0.676,	0.654
Corn flakes	0.024,	0.028
Flour		
White	0.187,	0.197
Whole wheat	0.627,	0.645
Noodles, egg	0.662,	0.583
Oat breakfast cereal, prepared	0.451,	0.406
Oats, quick	0.114,	0.105
Rice		
Polished	0.334,	0.303
Brown	0.383,	0.394
Rice breakfast cereal, puffed, prepared	0.026,	0.031
Wheat cereal	0.241,	0.241
Wheat breakfast cereal, prepared	0.110,	0.100
Mean excluding corn flakes and rice cereal	0.387	

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Table 28.

Selenium Content of Miscellaneous Products
(17)

Product	µg Se/gram	
Egg		
Yolk #1	0.174,	0.193
Yolk #2	0.192,	0.174
White #1	0.057,	0.057
White #2	0.046,	0.043
Saccharin	0.005,	0.005
Sugar		
Brown	0.012,	0.010
White	0.003,	0.003

Table 29.

Selenium Content of Dairy Products
(17)

Product	µg Se/gram	
Cheese		
American, processed	0.090,	0.090
Cottage	0.054,	0.050
Swiss	0.101,	0.108
Cream, table	0.005,	0.006
Cream substitute	0.034,	0.033
Milk		
Evaporated, canned	0.012,	0.013
Skim	0.045,	0.050
Skim, powdered, dried #1	0.098,	0.094
Skim, powdered, dried #2	0.243,	0.238
Whole homogenized	0.013,	0.011
Mean	0.069	

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Table 30.

Selenium Content of Meats and Seafoods
(17)

Product	µg Se/gram	
Meat		
Beef		
Round steak	0.363,	0.318
Ground	0.208,	0.189
Liver	0.454,	0.409
Kidney	1.41,	1.69
Pork		
Chop	0.217,	0.261
Kidney	1.89,	1.90
Lamb		
Chop	0.172,	0.184
Kidney	1.38,	1.47
Chicken		
Breast	0.106,	0.125
Leg	0.121,	0.151
Skin	0.154,	0.146
Mean excluding kidneys	0.224	
Seafoods		
Lobster tail	0.634,	0.681
Shrimp, shelled, deveined	0.572,	0.604
Cod, fillet	0.465,	0.390
Flounder, fillet	0.335,	0.338
Oysters	0.646,	0.659
Mean	0.532	

Table 31.

Selenium Content of Strained Baby Foods
(17)

Product	µg Se/gram	
Beef	0.118	0.113
Chicken	0.112,	0.101
Lamb	0.123,	0.138
Liver	0.247,	0.269
Pork	0.114,	0.133
Carrots	0.002,	0.002
Green beans	0.005,	0.004
Peaches	0.003,	0.004
Pears	0.002,	0.005
Oatmeal cereal with applesauce and bananas	0.026,	0.033
Rice cereal with applesauce and bananas	0.019,	0.023
Vanilla custard pudding	0.016,	0.015
Mean	0.068	

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B. Persistence and Degradation

The weathering of sedimentary rock is thought to have created seleniferous soil. The deposition of selenium in sediments may be a product of volcanic ash, which contains significant levels of selenium. Accumulation and deposition of selenium by marine organisms also contributed.[12,15] The cycling of selenium in nature is summarized in Figure 6.

The selenium in young soils occurs predominantly in an inorganic form not utilizable by crop plants. Selenium accumulator (indicator) plants and some bacteria can function as selenium converters, removing the selenium from the soil and returning it in an organic form, which can be absorbed by other plants. The established biological valence transformations of selenium are reduction reactions. Selenate, and selenite can be converted to elementary selenium and selenoorganics by plants and microorganisms. Evidence for microbial oxidation of selenium is the lack of buildup of organic selenium compounds in nonsterile soils. However, the oxidative stages in the selenium cycle may be abiotic, as about 80 per cent of elementary selenium as dust in moist air is converted to selenium dioxide, which reacts with water to form selenious acid. Selenite is the most unstable form of selenium, and may be readily oxidized to selenate.[12,15]

Organic selenium and some selenium salts are easily leached from soils, and the selenium content of irrigation waters, springs, and shallow pools may be quite high in seleniferous regions. In alkaline soils, much of the selenium may be trapped by reaction with iron oxides. Iron-selenium compounds are quite insoluble under basic conditions, and the selenium available to indicator plants and microorganisms, but not crop plants. Acidification of the soil by microbe action can cause the conversion of insoluble inorganic selenium to soluble forms.[12,15]

Selenium may enter animals in several ways. The predominant route is through ingestion of vegetation containing high levels of selenium. Carnivores obtain selenium by eating prey fed on seleniferous forage. Selenium

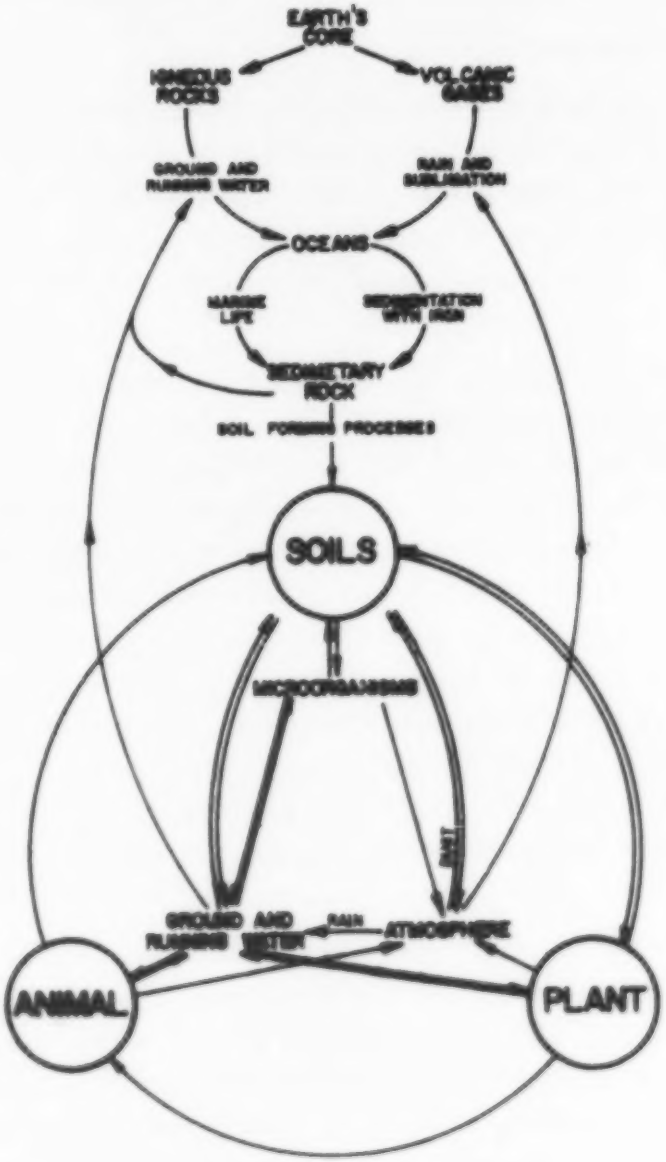

Figure 6

Cycling of excessive levels of selenium in nature

in drinking water is another possible source. Small amounts of selenium may be inhaled, as animals, plants, and microorganisms all produce volatile selenium compounds. Animals excrete selenium in the feces, urine, perspiration, as well as in the breath.

A feeding experiment in sheep was designed to determine the movement of physiological levels of selenium from the soil through plants into animals and their offspring using two groups of ewes fed alfalfa.(52) One group was fed alfalfa grown on untreated soil, and the other group ate alfalfa raised on selenium-treated soil. The physiogical effect on the animals was measured by examining the incidence of white muscle disease (WMD) in the offspring of both groups. No incidence of WMD was observed in the lambs of ewes fed the alfalfa grown in selenium-treated soil. (This alfalfa contained 2.6-2.7 ppm selenium compared with 0.01 to 0.04 ppm selenium in the untreated). In the other group, 25 per cent of the offspring died of WMD during the first six weeks of life. Almost 70 per cent of the lambs surviving to six weeks of age showed WMD lesions. The lambs in the former group had extremely higher levels of selenium in their tissues than the lambs with the high incidence of WMD.

The ewes fed on the high-selenium alfalfa were retained and maintained on low-selenium alfalfa (<0.01 ppm) for the next year. Of the lambs born to these ewes at the end of that time, only one out of thirteen surviving to six weeks of age exhibited symptoms of WMD. This was not evident until microscopic examination of the muscle was carried out. In a new group of ewes fed on the low-selenium alfalfa, over 50 per cent of the surviving six-week old lambs had WMD. The protection afforded the lambs of the former group of ewes even a year after the removal of selenium from the diet indicates a long retention time for the element. This experiment also illustrates soil retention of selenium in an indirect way as measured by selenium content of the alfalfa. The alfalfa from the selenium-treated plot (1 ppm selenium applied) contained 2.6 to 2.7 ppm selenium. Alfalfa grown the following year, without another selenium addition to the soil, contained 0.43 to 0.57 ppm selenium. Since the same strain of alfalfa was used in both years, environmental factors must have been responsible for the differences in selenium accumulation. Unfortunately, soil selenium levels were not determined.

C. Bioaccumulation

As discussed in Section VIII under absorption and excretion, higher plants tend to accumulate selenium from the soil, depending upon the species, form of selenium, and other environmental conditions. [12] Certain species of plants thrive on soils containing levels of selenium toxic to most other plants. These selenium indicator plants may accumulate thousands of parts per million selenium, absorbing both organic and inorganic forms. Secondary indicator plants, which grow well on both seleniferous and nonseleniferous soils, can accumulate several hundred ppm selenium without adverse effects. Crop plants accumulate selenium readily, depending upon the chemical form, but concentrations over 30 ppm are toxic to most plants.

Animals readily absorb inorganic and organic selenium through the small intestine.[12,28] Monogastric absorption is more efficient than ruminant. Most of the selenium (70 to 80 per cent) is quickly excreted in the urine, breath, perspiration, and bile. The remaining selenium becomes bound or incorporated into blood and tissue proteins and is only slowly eliminated. The offspring of parents maintained on very low selenium diets for long periods of time show severe deficiency symptoms unless selenium supplementations are given, although the parents do not exhibit illness. The experiment with sheep discussed in Section IXB illustrates that the effective retention time of selenium is at least a year in sheep.[52]

Fresh water plankton accumulate selenium. Large amounts of selenium were found in the zooplankton in Lake Michigan downwind of Chicago. The range of the selenium concentration was 0.1 to 1.2 ppm.[53]

X. TOXICITY

A. Humans

1. Occupational Exposures

Workers exposed to fine elemental selenium dust collect the dust in the upper nasal passages, producing catarrh, nose bleeding, and loss of smell. A few cases of selenium dermatitis have occurred. Exposure to fumes of elemental selenium produces frontal headache, intense irritation of the eyes and nasopharyngeal passages, slight difficulty in breathing, and uvular edema. Exposed workers recovered within three days and no ill effects persisted.(25)

Most of the workers in a Japanese factory making selenium rectifiers are minors and children. Hypochromic anemia and drastic leucopenia were prevalent in workers who had been employed in the factory for a long time. Fingernails were damaged from regular contact with selenium, and increasing numbers of female workers reported irregular menses or menostasis.(12)

Selenium dioxide readily dissolves in water producing selenious acid. Intense local irritation and inflammation of the skin and mucous membranes occur upon contact. Selenium dioxide, selenious acid, and selenite may be absorbed through the skin, resulting in internal accumulation. Allergic dermatitis to selenium dioxide may develop, especially in fairheaded people. Sodium thiosulfate solution is used to treat exposure.(25)

Hydrogen selenide fumes, from an etching and imprinting operation on a steel strip using a selenious acid bath induced nausea, vomiting, metallic taste in the mouth, extreme lassitude, and fatigability. Olfactory fatigue and garlic breath also occurred.(25)

Selenium oxychloride is a severe vesicant, capable of producing a third degree burn which is extremely painful and slow to heal.(25)

Dimethyl selenide produces acute sore throat and pneumonitis. A 13 year study of selenium intoxication in industrial workers found that the mortality rate was similar to that of the normal population. To date, no long-term systemic effects are known which can be attributed to selenium.(25)

2. Epidemiological Exposures

Acute dermatitis which proved to be selenium poisoning was observed in a rancher in South Dakota. Appreciable quantities of selenium were found in the water, meat, vegetables, and dairy products from his ranch.(12) The patient was treated with bromobenzene to eliminate selenium from his system.

Many other cases of selenium dermatitis have been reported from North Dakota, South Dakota, Montana, Wyoming, and Nebraska. Symptoms included various degrees of skin rash, follicular rashes over the hairy parts of the body, extreme fatigue, and dizziness. The selenium sources were home-grown farm products in the diet. Recovery was achieved by removal of selenium from the diet.(12)

Well water from the Wasatch geological formation in Utah contained 9 ppm selenium. Chronic selenosis occurred in the children, parents, and dog drinking the water. No selenium was found in the food. Lassitude, total or partial loss of hair, discoloration and loss of fingernails were symptoms of the condition. A halt in the use of the water brought regrowth of the hair and nails and increased mental alertness.(25)

People living in seleniferous regions should be conscious of the possible danger of selenium in foods and water.(12,25)

Chronic selenosis has been known in an area of Colombia, South America since the 1600's. The black "peladero" soil in the area contains extremely high levels of selenium, which accumulate in the corn and other crops, and forage plants and are present in the streams in the area. Loss of hair and nails, and the birth of malformed babies have been symptoms of the selenosis in Indians living in the area. Animals grazing there lost hair and hoofs, showed malformations of lips and legs, a number of animals aborted, and eggs failed to hatch.(12)

B. Mammals

1. Acute Toxicity

Injected doses of selenium compounds at levels of 200 micrograms selenium per kg body weight usually produce toxic effects in cattle, horses,

and pigs. Consumption of plant materials containing 400 to 800 ppm selenium proved fatal to sheep, hogs, and calves. Table 32 indicates minimum lethal doses of selenium compounds for both livestock and laboratory animals. Table 33 lists the LD_{50}'s for some intraperitoneally administered selenium compounds in the mouse and rat.

Symptoms of acute selenosis in livestock include uncertain gait and stance, rapid, weak pulse (90 to 300) elevated body temperature (103 to 105 F) labored respiration with mucous rales, bloody froth from the air passages, bloating accompanied by abdominal pain, and diuresis. The mucous membranes are pale or bluish, and the pupils dilated. Lethargy and prostration precede death. Death is due to respiratory failure. The urine may contain 0.1 to 20 ppm, and the bile 1 to 6 ppm selenium. Highest selenium concentrations are found in the liver (22 ppm), blood (5 to 15 ppm), kidney (10 ppm), spleen (8 ppm), and brain.(12)

Gross and microscopic pathology showed evident systemic damage, especially hemorrhage, congestion and necrosis of the parenchymatous organs.(12)

Acute selenite selenosis in laboratory animals was manifested by initial signs of nervousness and fear, vomiting, diarrhea, and respiratory difficulty. Quietness and somnolence were followed by opisthotonos, tetanic spasms in the muscles of the extremities, clonic spasm, and death. The heart rate remained normal, and a gradual fall in blood pressure began 15 to 30 minutes after injection and continued until death.(12)

2. Subacute and Chronic Poisoning

a. The blind-staggers type of chronic selenosis appears in cattle ingesting large amounts of indicator plants, which contain a moderate or high concentration of water-soluble organic selenium. Severity of the toxicity depends upon the type of plant ingested, stage of growth, and soil type. In the first stages of poisoning, the animal may walk in circles, stumble over objects in his path and exhibit little desire to eat or drink. In the second stage, the manifestations of the first stage become more severe, and the front legs become weak and seem unable to support the animal. Loss of desire to eat or drink occurs, although no paralysis is exhibited by the tongue and throat

muscles involved in swallowing. The final stage prior to death is a paralytic stage. The tongue and throat muscles are paralyzed. The animal is nearly blind, respiration is labored and accelerated, and there is evidence of great abdominal pain, which causes constant grating of the teeth and salivation. A subnormal body temperature, swollen and inflamed eyelids, and cloudy corneas are evident. The mucous membrane of the mouth is pale. The animal may appear emaciated. The onset of the third stage is sudden, and death usually occurs within a few hours.

Impaction of the rumen, and stasis in the omasum are pathological findings characteristic of blind staggers. If the syndrome is recognized during the first or second stage, some relief may be afforded by drenching the animal with warm water. Small doses of strychnine sulfate may be injected. No laxative should be administered during the initial stages of treatment to minimize the irritation to the stomach.

b. Chronic poisoning of the alkali disease type results from the ingestion of feedstuffs (corn, wheat, barley, oats, grasses and hay) containing 10 to 30 ppm selenium. The selenium is predominantly present in the proteins of these feeds. The amount of selenium is related to the availability of soil selenium.

The general symptoms of alkali disease include lack of vitality, anemia, stiffness of joints, lameness, roughened coat, loss of hair, and hoof lesions and deformities. Death may occur within two months after a horse is placed on a seleniferous pasture.

In alkali disease higher concentrations of selenium appear in the hair and hoofs than the other parts of the body. The heart and liver exhibit the most damage. The heart becomes soft, flabby and atrophied. Fibrosis is evident in the liver and kidneys.

The only certain treatment is to discontinue feeding seleniferous grain and forage.[12]

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Table 32.

Minimum Lethal Dose of Selenium Compounds
(12)

Selenium Compound	Mode of Administration	Animal	mg Se/kg
Selenite	oral	horse, mule	3.3
Selenite	oral	cattle	3
Selenite	subcutaneous	pig	1.2 (5 days)
Sodium selenite	intraperitoneal	rat	3.25 - 3.5
Sodium selenate	intraperitoneal	rat	5.25 - 5.75
Sodium selenite	intravenous	rat	3.0
Sodium selenate	intravenous	rat	3.0
Sodium selenite	intravenous	rabbit	1.5
Sodium selenate	intravenous	rabbit	2.0 - 2.5
Colloidal selenium	intravenous	rat	6.0
l-Selenocystine	intraperitoneal	rat	4.0
Selenomethionine	intraperitoneal	rat	4.25
d, l-selenocystine	intraperitoneal	rat	20 - 25
beta-seleninopropionic acid	intraperitoneal	rat	25 - 30
beta, beta-prime-diseleno-diproprionic acid	intraperitoneal	rat	25 - 30
beta-selenodipropionic acid	intraperitoneal	rat	40

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Table 33.

LD_{50} for Some Selenium Compounds
Administered by Intraperitoneal Injection
(12)

Selenium Compound	Animal	LD_{50} (mg Se/kg)
isoselenourea sulfate	rat	3.0 ± 0.3
selenohomocystine	rat	3.5 ± 0.4
d,-l, or d,l-meso-selenocystine	rat	4.0 ± 0.2
selenomethionine	rat	4.5 ± 0.3
selenotetraglutathione	rat	6.0 ± 0.3
dimethyl selenide	mouse	1300
dimethyl selenide	rat	1600
6-selenopurine	mouse	160 ± 37

c. Chronic Selenosis by Inorganic Selenium

Selenite administration produced symptoms similar to those seen in alkali disease. Inappetence was the most pronounced symptom. Some lesions characteristic of alkali disease were produced in equines and pigs after repeated small doses of selenite. In equines emaciation, drowsiness, and loss of appetite preceded death; loss of hair and separation of the hoof occurred in pigs. Peculiar trembling of the skeletal muscles, shivering during exercise, anorexia and inability to rise were noted in cattle.(12)

d. Subacute and Chronic Selenium Toxicity in Laboratory Animals(12)

Considerable species and individual variation to the toxic effects of selenium exists. In subacute and acute selenosis, when fractions of a minimum lethal dose are given, the effect is cumulative. A continued increase in the manifestations of toxicity upon repeated administrations suggests that no altered susceptibility or acquired tolerance develops. Young animals are more susceptible to selenium intoxication than older animals; female rats are more susceptible than males. Rats are apparently able to distinguish between various concentrations of selenium in the diet even when the increments were small.

In general, concentrations in diets of less than 5 ppm selenium as seleniferous grain prevented normal growth, 9 ppm resulted in death of young animals, and 10 ppm produced restricted food intake in adult rats. Food which contains 40 to 50 ppm of selenium even when consumed in small amounts is more toxic than food which contains 10 to 12 grams per day.

The toxic manifestations of inorganic selenium compounds and seleniferous grains are almost identical under similar experimental conditions. The addition of 7.2 ppm selenium as seleniferous wheat or 10 ppm as selenite to the diet was toxic to dogs.

Chronic selenosis may be produced in various experimental animals with concentrations of 10 to 16 ppm of selenium in the diet or drinking water. If the selenium concentration is between 20 and 30 ppm, subacute selenosis may develop. Intraperitoneal, oral or subcutaneous administration of selenite or selenate produces chronic or subacute selenosis in rats when the dose is 1.5 to 2.2 mg per kg body weight.

Subacute and chronic selenosis are characterized by marked loss of body weight, anorexia followed by cachexia, ascites and edema, anemia, intermittent vomitting and decreased hepatic function.

3. Teratogenicity

Placental transmission of selenium has been shown to occur. Offspring of selenized parents tended to be underweight, and malformations were evident in lambs born to ewes grazed on seleniferous pastures. Congenital alkali disease appeared in a 14-day-old colt born of a mare in which symptoms appeared during gestation.(35)

Gross abnormalities were observed in chick embryos from eggs laid by selenium-fed hens. On farms in seleniferous regions the failure of eggs to hatch may be due to deformities which prevent hatching, and not to infertility. Deformities in embryos were produced by injecting eggs with selenite before incubation.(54)

4. Carcinogenicity

Conflicting data has been presented on the role of selenium as a carcinogen.(55) Three studies exist which implicate the carcinogenic nature of selenium, but it has been argued that low-protein diets, the inability of the liver tumors to metastasize and the lack of controls in one study render the data inconclusive. In the most recent study young rats were given 20 ppm selenate, selenite and tellurite in drinking water. The selenite produced markedly toxic effects, particularly in the males. The selenate level was increased to 3 ppm at the end of one year, and the experiment continued for the lifetime of the rats. At death, 42 per cent of the selenate-fed rats exhibited benign or malignant tumors compared with 17 per cent of the controls, 18 per cent in the tellurite-fed animals, and 13 per cent in the selenite-fed

ones. All of the tumors in the controls and 91 per cent in the selenate-fed rats developed after two years. (Selenite-fed rats were all sacrificed by the end of 23 months because of the high mortality and toxicity of the compound.)[55]

5. Anticarcinogenicity

A growing number of apparent antitumor actions of selenium compounds have appeared.[56,57] Male rats receiving m'-methyl-p-dimethyl-aminoazobenzene (m'-DAB) had an incidence of 93 per cent liver tumors compared with 31 per cent present in animals also receiving 5 ppm selenium as sodium selenite.

Intraperitoneal injection of selenocystine (1 mg/kg) into rats injected with Murphy lymphosarcoma cells inhibited tumor growth. Rats not receiving selenocystine treatment had tumors approximately 30 cm^2 in area after 21 days compared with tumors 8 cm^2 in rats receiving selenocystine. The inhibition was quite specific to selenocystine, since benzyl-selenium alanine did not inhibit tumor growth.

In human patients, two with acute leukemia and two with chronic myeloid leukemia, daily doses of 100 mg selenocystine administered in capsules for 10 to 57 days produced a marked reduction in the immature leukocytes circulating in the blood. Therapy had to be discontinued despite promising effects because the patients experienced nausea and vomiting.

The addition of sodium selenide to croton oil, which was painted on to the skin of mice, reduced tumor incidence from 132 tumors in mice painted without selenide to nine tumors.

In mice, 6-selenopurine was as effective as 6-mercaptopurine in inhibiting the growth of lymphomas. Intraperitoneal 2-amino-6-selenopurine also inhibited tumor growth, but oral doses were ineffective.

A very high negative correlation between blood selenium levels in human males and "human cancer death rates" has been reported. ($R = 0.96$; $P<0.001$). Studies with microorganisms indicate that selenium interferes with cell division processes.[12] More complete studies are necessary to ascertain the exact interactions of selenium compounds.[25]

6. Factors Affecting Selenium Toxicity

Arsenite and arsenate, but not arsenic sulfides, are equally effective in preventing the toxic action of selenium in seleniferous wheat, sodium selenite and selenocystine. Organic arsenicals have been found to give partial protection against chronic selenosis in rats. Arsenic increases biliary excretion of selenium.(55)

The presence of sulfide in an injection to rats reduces the toxic effects of selenite. Dietary sulfate reduced selenium-produced growth depression, but not liver damage. Sulfate modified chronic selenosis produced by selenate, was less effective against selenite, and ineffective against seleniferous wheat.(12)

Increasing the protein content of the diet affords protection against both organic and inorganic selenium poisoning. Toxic symptoms and high mortality occurred in rats fed a diet containing 10 ppm selenium as seleniferous wheat and 10 per cent protein. Scarcely any pathological effects occurred when the same diet contained 30 per cent protein. Casein, lactalbumin, ovalbumin, wheat protein, dried brewer's yeast, and desiccated liver gave effective protection against chronic selenosis; gelatin and edestin gave only slight protection.(12)

Glucosamine, linseed oil, homocystine, creatine, methionine, choline, and betaine afford partial protection against chronic selenosis under some conditions. High fat diets also provide some protection. Germanium and antimony are also partially protective. Zinc enhances the effects of selenium.(12)

Bromobenzene has been used to treat selenium poisoning. Theoretically, bromobenzene reduces selenium toxicity by increasing urinary excretion as the selenium analog of mercaptohippuric acid, the normal excretory product of bromobenzene. The effect of bromobenzene on selenium excretion is quite variable. Its innate toxicity raises questions about its use as a detoxicant.(12)

Two, three-dimercaptopropanol (BAL) has an additive toxic effect in chronic selenosis in rats, but liver damage was absent. BAL did not block the beneficial effect of arsenic on selenium toxicity.(12)

C. Fish

Selenium poisoning has been produced in goldfish and catfish. The catfish died within 48 hours after receiving a single intraperitoneal injection of 0.15 mg or more selenium as sodium selenite. An injection of 0.05 mg selenium produced no immediate effects, but exophthalmus, ascites, tissue edema, and death occurred within 12 to 15 days. Marked reductions in blood specific gravity, red blood cells, and hemoglobin were noted.(12)

D. Invertebrates

Selenium has been effectively used to control aphids, mites, and red spiders in greenhouses. Surprisingly, larvae and adults of some species of insects are apparently immune to selenium poisoning. Larvae of Cecanthoscelides fraterculus, a bruchid, and Bruchophagus mexicanus, a seed chalcid, were capable of consuming seeds of Astragalus bisulcatus containing 1475 ppm selenium. The insect bodies contained 67 ppm selenium. Flies containing 20 ppm selenium, fly larvae, containing 7.5 to 10 ppm selenium, were found on Astragalus pectinatus, whose roots contained 190 to 420 ppm selenium. Grasshoppers and blister beetles have also been found feeding on high-selenium plants.(12)

Soil and leaf nematodes were effectively controlled by sodium selenate. Selenium applications to soil may pose real health hazards to humans and farm animals if used on garden or forage crops.(12)

E. Plants

Organic selenium (as Astragalus extract) produced less growth inhibition and toxicity to crop plants than inorganic selenium. In order of diminishing toxicity, some selenium compounds injurious to plants are: selenious acid, selenic acid, selenite, selenate, and selenocyanate.(12)

Selenate toxicity in crop plants appears as white chlorosis of the leaves. Older leaves become yellowed. The roots become stunted, but are otherwise normal in appearance.

Leaves injured by selenite are likely to be darker green than usual. Older leaves wither and become brown. Extremely high concentrations of selenite induced white chlorosis and premature death of wheat plants. Roots poisoned

by selenite have a pinkish appearance, which, upon microscopic examination, is found to be due to red granules within the roots. The red granules are thought to be elemental selenium. The main roots are thickened, with suppressed development of lateral roots.(12)

F. Microorganisms

Although selenium inhibits growth of many microorganisms, some strains of bacteria apparently can adapt to a high-selenium environment. Escherichia coli grown in a medium containing 2×10^{-4} molar selenate exhibits a lag in growth lasting for 24 to 48 hours. After that time, the growth ultimately attained the same levels as control cultures. The adapted E. coli cultures maintained their resistance to selenate even after nine transfers on selenate-free nutrient agar plants.(58)

Cultures of bacteria isolated from selenium-rich soils in Russia were more resistant to high concentrations of the element than cultures isolated from selenium-depleted soils. A Moscow-region strain of Bacillus megaterium could be acclimated to high selenium by growth on a medium containing 0.05 ppm selenium.(59)

G. Results of Personal Contacts with Medical Personnel

A total of 74 toxicologists and medical examiners throughout the United States were contacted by telephone or letter with regard to their professional acquaintance with incidences of accidental poisoning of humans attributed to selenium or its compounds. Of the 31 responses, there were five reported instances of selenium poisoning, none of which were fatal. These are described as follows:

3 cases - accidental poisoning by "Selsun" dandruff treatment preparation (three widely separated instances; two children, one adult). Most serious symptom was alopecia.

2 cases - accidental poisoning of two families (parents and children) in Alabama due to high selenium content of well water (8 and 12 ppm). Symptoms included skin lesions.

XI. CURRENT REGULATIONS

From public water supplies the permissible criterion for selenium (2^+,4^+,6^+) is 0.01 mg/l (10 ppb).[60]

The State of Illinois has established the regulation for public water that the "maximum allowable twelve-month average selenium concentration" is 0.01 mg/l. No single analysis shall show a concentration at any time of more than 1.5 times this value. The justification given for this criterion is is that "to maintain the rather narrow margin of safety imposed by the background ingestion of selenium from food, drinking water must not contain more than 0.01 mg/l of selenium".[61]

A tentative allowable concentration of 1 mg/l of selenium in urine was recommended for rural populations living on seleniferous soil and for workers exposed to selenium or its compounds.[20]

American Feed Manufacturers Association, Inc., sent a petition to the Food and Drug Administration to amend the Food Additive Regulations to permit the safe use of selenium as a nutrient in the feed of chickens, turkeys, and swine. The proposal provides that selenium as sodium selenite or selenate may be added at a level not to exceed 0.1 ppm for chickens and swine and 0.2 ppm for turkeys. The status of the petition is dependent on the comments arising from the publication of the proposed changes.[62]

XII. STANDARDS[63]

Threshold Limit Values elementary selenium and its common inorganic compounds:	0.2 mg (as Se)/m^3
U.S.S.R.:TLV (1959)	0.1 mg/m^3
TLV for Hydrogen Selenide (H_2Se)	0.05 ppm (equivalent to approximately 0.2 mg/m^3)
TLG for Selenium hexafluoride (SeF_6)	0.05 ppm or approximately 0.4 mg/m^3

XIII. SUMMARY AND CONCLUSIONS

A. Summary

Selenium is a paradoxical element, beneficial or essential in amounts varying from trace to parts per million concentrations for humans, animals and plants and quite toxic to animals at concentrations which may exist in the environment. Also, the sensitivity to selenium and its compounds is extremely variable in all classes.

Selenium production is a by-product of copper recovery both in the U.S. and elsewhere. The United States has slipped from its position as the world's leading selenium producer as a result of the 1967-1968 copper strikes. However, the production of selenium does not present any economic or strategic problems. At present, selenium imports amount to less than one-half of the production, and less than sixty per cent of the selenium available to U.S. copper refiners is recovered. In practically all the applications for selenium, the chemically similar compounds of sulfur and tellurium can be substituted. However, only selenium is suitable in reusable photosensitive plates considering current technology.

Selenium is widely distributed in nature, but is found in greater concentrations associated with sulfide minerals of lead, iron, copper and other metals. The concentration is certain "seleniferous" soils is great enough that specific species of plants growing there pick up sufficient selenium to be toxic to animals. This natural source of selenium entering the environment is significant but less than that from the weathering of natural rock.

Selenium entering the environment from activities of man is estimated at 3,500 metric tons per year, of which most is attributed to the combustion of coal. These contributions appear to be small in comparison to contributions from natural sources.

Selenium and its compounds are only moderately toxic to man. The effects disappear when the exposure ceases. Individual sensitivity seems to be a big factor which is highlighted by instances of dermatitis among workers handling selenium. A number of cases of accidental human poisoning by selenium in well water and by selenium sulfide (dandruff treatment) have been reported.

Selenium compounds are absorbed through the small intestine and excreted in the urine, feces, exhaled air and perspiration. Inorganic selenium salts become tightly bound to protein, binding with free sulfhydryl groups. Organoselenium compounds are metabolized like their sulfur analogs. Tracer studies with ^{75}selenium amino acids, for example, have made use of this property. Selenium is a growth requirement in livestock, poultry and rodents. White muscle disease in sheep and exudative diasthesis in chickens are selenium deficiency diseases prevalent in areas of the world with low-selenium soils.

Plants are extremely efficient accumulators of selenium especially organoselenium, but tolerance of plants to selenium varies greatly. Selenium "indicator" plants may accumulate thousands of ppm selenium without ill effects. and in these plants, selenium is a growth factor. The selenium is present in small molecular weight organic compounds. Although crop plants accumulate selenium, concentrations of 25 to 50 ppm may produce phytotoxicity. Selenium is incorporated into protein in crop plants, but selenium does not appear to be a necessary growth factor.

High selenium plants and waters are significant dangers to livestock in seleniferous zones due to the extreme toxicity of the element. Chronic or acute blind staggers or alkali disease may occur depending upon the type of plant and amount ingested. Urinary selenium levels appear higher in humans ingesting foods raised in seleniferous areas, and chronic and acute cases of poisoning have been reported.

Selenium is transmitted from the mother to the fetus. Reduced reproduction rates and weakened offspring occur in selenium-deficient mothers. Excessive selenium may act as a teratogen. The majority of the evidence indicates that selenium compounds can function as antitumor agents rather than carcinogens. Selenium compounds function as growth inhibitors in many microorganisms. Animals, plants and microorganisms reduce selenium, but metabolic oxidation has not been clearly established in any species.

B. Conclusions

The following conclusions are based on the information contained in this report:

(1) The major hazard to man from selenium arises from habitation

of regions of the U.S. in which seleniferous soils exist. Dietary and water intake high in selenium can produce severe toxic effects. Mild to moderate occupational hazards from selenium also exist.

(2) Sources of selenium attributal to the activities of man are small in comparison to the natural sources (soil, plants, rock). However, localized high concentrations may result from dumping of solid wastes from coal and fuel oil combustion, fuel desulfurization, and from mining and processing of a number of metallic ores.

(3) Hazards to plants and livestock from selenium exist in areas of seleniferous soils, but these are generally well-known and controlled.

C. Recommendations

The following recommendation is based on the summary and conclusions presented above:

(1) The amounts and fate of selenium entering the environment through solid waste disposal from various activities of man should be more closely studied. These solids arise from the desulfurization and combustion of selenium-containing fuels (coal and fuel oil) and from various mining activities.

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